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ARCHIVES OF SURGERY

VOLUME 17 SEPTEMBER, 1928 NUMBER 3

SIMPLE, NONSPECIFIC ULCER OF THE COLON *

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BOSTON

Simple, nonspecific ulcer is a pathologic lesion which occurs in the colon. It may exist as a single lesion or it may be multiple. The fact that it does occur and usually tends to perforate should make one ever mindful of its occurrence.

By simple ulcer is meant an ulcerating lesion which is not due to the action of any specific organism such as the specific ulcer of tuberculosis, syphilis, ulcerative colitis, dysentery, typhoid fever or to the local action of any chemical agent, and which is not secondary or above a malignant tumor causing constriction.

This series comprises fifty-three cases; fifty were collected from the literature and three are cases that I observed. In this series, the cases include simple ulcers of the colon regardless of their location. Both perforated ulcers and chronic latent ulcers have been considered, since in certain patients the ulcers may have been multiple, one ulcer having perforated and the others remaining quiescent. I have included also simple ulcer of the rectum as I believe the etiology and pathology of this disease to be the same as that of simple ulcer on any segment of the colon. I have also included those simple ulcers which, in the process of healing, have caused a stenosing cicatrix, and thus given rise to partial or complete obstruction. In other words, all ulcers of the colon of a nonspecific nature that were studied by autopsy, resection or visualization at time of operation were included.

HISTORICAL

There is nothing in the American literature regarding this lesion aside from a case report by Lyle,[1] in 1912, and a report of three cases by Dickinson,[2] in 1922, entitled, "Perforating Ulcers of the Cecum." Most of the cases reported are found in the French and German literature.

This disease is not new, and was first described by Cruveilhier [3] in 1830. Since then, there have been many cases reported. No attempt

* From Surgical Department of Beth Israel Hospital, Boston, Mass.

1. Lyle, H. H. M.: Simple Ulcer of the Colon: Perforation, Ann. Surg. **56**:473, 1912.

2. Dickinson, G. K.: Perforating Ulcers of the Cecum, J. A. M. A. **78**:1792 (June 10) 1922.

3. Cruveilhier, J.: Rectum, Atlas d'anatomie et pathologie, 1830-1832, p. 4.

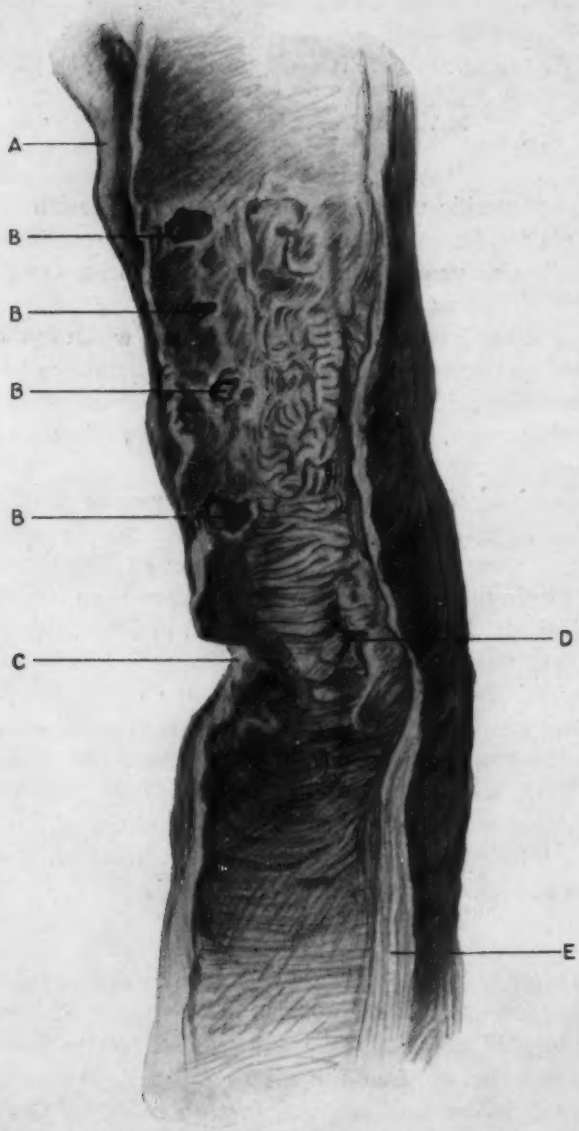

Fig. 1.—Postmortem specimen. *A* indicates the jejunum, adherent to surface of bowel; *B*, four smaller ulcers; *C*, the perforated ulcer; *D*, the narrowing and constriction of lumen, and *E*, marked hypertrophy of wall of colon proximal to ulceration and narrowing of lumen.

was made to collect a group of these cases for study until 1902. At that time, Quenu and Duval [4] reviewed twenty-seven cases of simple ulcer of the colon reported in the literature, including their own case which presented an ulcer of the rectum. Twenty-five of these patients were studied both in life and at postmortem examinations. Autopsy was incomplete in one case and in another case a histologic and gross study of the resected lesion was made.

In 1920, R. Soupault [5] reviewed twenty-seven cases of perforated ulcers of the colon. In his series, he eliminated ulcers of the rectum, ulcers situated above a stricture, ulcers causing stricture, though the strictures be due to a healed ulcer, and latent ulcers that have not perforated. Since then there have been several cases reported both in the French and German literature.

A brief historical résumé of the cases reported since 1837 is given in the following table.

REPORT OF AUTHOR'S CASES

CASE 1.—*History.*—Mr. S. B., a carpenter, aged 55, entered the surgical service of the Beth Israel Hospital on Nov. 4, 1926, with a complaint of pain in right lower quadrant. His family and past history were essentially negative. About two months previously, he had had an attack of pain in right lower quadrant which was not sharp or radiating. At that time, it was not severe enough to keep him in bed. The pain lasted one and a half days and was not associated with nausea or vomiting. The day before entrance to the hospital, he had a similar attack, but much more severe. Pain was localized just to the right of McBurney's point, causing him to double up. He was unable to work at this time. At no time previous to this history had he complained or consulted a physician for any abdominal condition. He was markedly constipated, always using cathartics. There never had been any nausea or vomiting. His stools varied in shape, and he described the color as being like that of weak tea. For two years he had had epigastric distress after meals. He weighed 171 pounds (77.6 Kg.). He had never been jaundiced.

Physical Examination.—The patient was a well developed and well nourished, middle-aged man, lying in bed with no objective signs of pain. The skin was moist and the face flushed. There was nothing else of note except marked tenderness of the whole right side of the abdomen. The maximum tenderness seemed to be in a line slightly above McBurney's point on the level with the umbilicus. There was a slight spasm in this region. The costovertebral angles were not tender. The abdomen was tympanitic throughout. No definite mass was made out over the tender area. A provisional diagnosis of acute appendicitis was made, and immediate operation was advised. The results of the urinalysis were negative. Examination showed: white blood cells, 12,300; temperature, 100 F.; the pulse rate, 100, and respiration, 22.

Operation.—Nov. 4, 1926: A right rectus incision was made under ether anesthesia, and a small amount of free fluid escaped from the peritoneal cavity. A

4. Quenu, E., and Duval, P.: Simple Ulcer of the Large Intestine, Rev. de chir. **26**:692, 792, 1902.

5. Soupault, R.: Contribution to the Study of the Perforation of Simple Ulcer of the Colon, Rev. de chir. **58**:480, 1920.

TABLE 1.—*Résumé of Cases Reported Since 1837*

Author	Sex	Age	Preoperative Diagnosis	Location	Perforation	Result
1. Marchesseaux: Bull. Soc. anat. de Paris **12**: 343, 1837	F	45	Intestinal perforation or peritonitis	Rectum	Perforation	Autopsy
2. Rogee: Bull. Soc. anat. de Paris **13**: 181, 1838	F	61	No diagnosis	Rectum	Perforation	Autopsy
3. Greenhow: Path. Soc. Newcastle & Calestead, Dec., 1848	M	72	No diagnosis	Splenic flexure	Perforation	Autopsy
4. Lebert: Traité d'anatomie et pathologie, générale et speciale, 1861, vol. 2, p. 206	M	47	No diagnosis	Splenic flexure	Perforation	Autopsy
5. Idem	M	47	Intestinal obstruction	Sigmoid	No perforation	Autopsy
6. Dujardine: Soc. de sc. méd. et Naturelle de Bruxelles, 1864	M	No age	Tumor of spleen, colitis	Splenic flexure	Perforation	Autopsy
7. Claus: Inaug. Diss., Zurich, 1856	M	47	Peritonitis	Splenic flexure	Perforation	Autopsy
8. Dickinson: Tr. Path. Soc. London **18**: 101, 1867	F	42	Peritonitis	Sigmoid	Perforation	Autopsy
9. Idem	M	42	Renal disease	Descending colon	Perforation	Autopsy
10. Robin: Thèse, Paris, 1869	M	66	No diagnosis	Descending colon	No perforation	Autopsy
11. Costin: Brit. M. J. **2**: 247, 1870	M	45	No diagnosis	Ascending colon	Perforation	Autopsy
12. Lepine: Bull. Soc. anat. de Paris **45**: 27, 1870	F	70	No diagnosis	Splenic flexure	No perforation	Autopsy
13. Chouppe: Bull. Soc. anat. de Paris **45**: 322, 1870	M	32	No diagnosis	Hepatic flexure	Perforation	Autopsy
14. Demange: Rev. méd. de l'est, Aug., 1897, p. 500	M	72	No diagnosis	Sigmoid	Perforation	Antopsy
15. Mickle: Brit. M. J. **1**: 637, 1883	M	45	Peritonitis and perforation	Sigmoid	Perforation	Autopsy
16. Bradbury: Lancet **2**: 637, 1884	M	30	No diagnosis	Cecum	No perforation	Autopsy
17. Oliver: Lancet **1**: 424, 1885	No age or sex		No diagnosis	Ascending colon	Perforation	Autopsy
18. Parker: St. Barth. Hosp. Rep. **23**: 213, 1887	F	18	Perityphilitis	Ascending colon	Perforation	Autopsy
19. Idem	F	49	No diagnosis	Ascending colon and cecum	Perforation	Autopsy
20. Ollier: Loire méd. **11**: 3, 1892	M	56	Peritonitis	Cecum	Perforation	Autopsy
21. Morel and Scheyron: Soc. de méd. de Toulouse, 1895	M	46	Cancer of cecum	Cecum	No perforation	Examination resected tissue, cured
22. Southam: Brit. M. J. **1**: 254, 1895	M	67	No diagnosis	Cecum	Perforation	Autopsy
23. Guinard: Thesis, Paris, 1896, no. 324	M	27	No diagnosis	Hepatic flexure	Perforation	Autopsy
24. Morel and Rispail: Soc. de méd. de Toulouse, 1896	M	67	Intestinal obstruction	Sigmoid	Perforation	Autopsy
25. Gamgee: Brit. M. J. **1**: 950, 1898	M	28	No diagnosis	Sigmoid	Perforation	No autopsy or pathologic examination, cured
26. Quenu and Duval: Rev. de chir. **26**: 692, 792, 1902	F	50	Ulcer of rectum	Rectum	No perforation	No autopsy, cured
27. Tedenat, quoted by Quenu and Duval	M	40	Paracecal abscess	Ascending colon	Perforation	No autopsy, cured
28. Idem	M	52	No diagnosis	Cecum	Perforation	Incomplete autopsy
29. Idem	M	68	No diagnosis	Ascending colon	Perforation	No autopsy, cured
30. Zickler: Beitr. z. klin. Chir. **67**: 189, 1910	F	25	Appendicitis	Ascending colon	Perforation	No autopsy, cured

TABLE 1.—*Résumé of Cases Reported Since 1837—Continued*

Author	Sex	Age	Preoperative Diagnosis	Location	Perforation	Result
31. Lyle: Ann. Surg. **56**: 473, 1912	F	29	Appendicitis	Ascending colon	Perforation	No autopsy, cured
32. Eunike: Deutsche Ztschr. f. Chir. **147**: 284, 1918	M	53	Strangulated hernia	Hepatic flexure	Perforation	Autopsy,
33. Soupault: Rev. de chir. **58**: 480, 1920	F	22	Appendicitis	Ascending colon	Perforation	No autopsy, cured
34. Bazy: Bull. et mém. Soc. de chir. de Paris **46**: 416, 1920	M	65	Obstruction, cancer	Ascending colon	Perforation	Autopsy
35. Idem	M	20	Appendicitis	Cecum	Perforation	No autopsy, cured
36. Gregoire: Bull. et mém. Soc. de chir. de Paris **46**: 420, 1920	M	55	Appendicitis	Sigmoid	Perforation	No autopsy, death
37. Sieur: Bull. et mém. Soc. de chir. de Paris **46**: 421, 1920	M	30	No diagnosis	Cecum	Perforation	Autopsy
38. Lardennois: Bull. et mém. Soc. de chir. de Paris **46**: 447, 1920	M	52	Peritonitis	Cecum	Perforation	Autopsy
39. Schoemaker, quoted by Soupault	M	No age	Peritonitis	Sigmoid	Perforation	Autopsy
40. Levy: Deutsche Ztschr. f. Chir. **165**: 366, 1921	M	No age	Appendicitis	Cecum	No perforation	No autopsy, cured
41. Ecot and Richard: Bull. et mém. Soc. anat. de Paris **92**: 138, 1922	No age or sex		Obstruction	Ascending colon	Perforation	No autopsy, cured
42. Dickinson: J. A. M. A. **78**: 1792 (June 10) 1922	M	No age	Appendicitis	Cecum	Perforation	Autopsy
43. Idem	M	22	Chronic appendicitis, chronic cholecystitis	Cecum	Perforation	Autopsy
44. Idem	M	56	Appendicitis	Cecum	Perforation	No autopsy, cured
45. Bowen: Guy's Hosp. Rep. **72**: 441, 1922	F	No age	Intestinal perforation	Sigmoid	Perforation	No autopsy, death
46. Idem	F	27	No diagnosis	Cecum	Perforation	No autopsy, does not state
47. Chalier and Mallet-Guy: Arch. d. mal de l'app. digestif. **13**: 521, 1923	M	50	Tuberculous peritonitis, tuberculous enteritis	Ascending colon	No perforation	Autopsy
48. Mintz: Zentralbl. f. Chir. **50**: 1208, 1923	F	18	Intussusception	Cecum	No perforation	Resection, pathologic examination, cured
49. Broere: Nedrl. Tijdschr. v. Geneesk. **67**: 2274, 1923	F	80	No diagnosis	Sigmoid	Perforation	Autopsy
50. Boss: Beitr. z. klin. chir. **140**: 40, 1927	M	38	No diagnosis	Sigmoid	Perforation	No autopsy, cured
51. Barron: In the text.....	M	56	Appendicitis, malignancy	Ascending colon	Perforation	Autopsy
52. Barron: In the text.....	F	23	Appendicitis	Cecum	No perforation	No autopsy, cured
53. Barron: In the text.....	F	32	Simple ulcer of rectum	Rectum	No perforation	No autopsy, cured

large, inflammatory mass was found at the hepatic flexure, firmly tied down to all the adjacent structures. The mass was covered by omentum. The distal portion of the ascending colon seemed to be adherent to the transverse colon. The adhesions were separated without much difficulty, and a small opening was found in this inflammatory mass from which thick, greenish pus escaped. The appendix was retrocecal and did not show evidence of acute inflammation. It was removed in the usual manner. The abdomen was closed in layers with two cigaret wicks.

The patient made an uneventful convalescence. There was a purulent discharge from the abdominal sinus which eventually cleared up, and the patient was discharged well, eighteen days after operation. The wound was well healed throughout. The patient was advised to come back for further x-ray study, but he did not return to the hospital until Dec. 14, 1926.

The following report was made at this time from the x-ray department: The tone of the stomach was good, and peristalsis was normal; the outline of the stomach was regular while the duodenum showed constant irregularity. Examination made five hours after the ingestion of the barium meal showed a small residue in the stomach (from 10 to 15 per cent). The head of the meal was in the distal portion of the colon (hypermotility). Examination made twenty-four hours after the barium meal showed the head of the meal in the ampulla of the rectum. The entire colon was empty. Examination of the colon with an opaque enema did not show any obstructive lesions. The cecum and ascending portion of colon showed finger-like defects, but its outline was rather smooth. The terminal ileum was well filled, distended, and also showed irregular filling. The inferior border of the transverse portion of colon was slightly irregular and appeared to be fixed.

The hypermotility of the colon when examined five hours after the barium meal and also the rapid emptying of the colon after the twenty-four hour examination suggested marked colonic irritation. This, together with the filling defect in the terminal ileum, cecum and ascending portion of the colon, definitely pointed to a pathologic process in that region. The entire picture was not characteristic of a malignant condition. There was a possibility of a tuberculous lesion.

The patient did not appear again until his next admission to the hospital on Aug. 15, 1927. This was the first time that he came to my attention. Again, the chief complaint was pain in right side of the abdomen. The patient said that he had been operated on at the Beth Israel Hospital seven months before. He had been discharged as cured, but he had never felt well since his operation. The week before his second admission, the pain was rather severe and stabbing, and seemed to be getting worse. The family physician said that he had felt a tumor in the patient's right side since he left the hospital, and that the tumor was now growing larger. His appetite was good. During the past four days, he had been constipated, requiring enemas. Previous to this, he said that he had had bowel movements about eight times a day; the stools were watery and accompanied by considerable gas. There had not been any nausea or vomiting. His best weight was 210 pounds (95.2 Kg.) one year before admission to the hospital. At the time of the previous operation, he weighed 171 pounds (77.6 Kg.); his present weight was 164 pounds (74.4 Kg.).

On his discharge from the hospital after his previous operation, he gained in weight, but recently he had been losing. His past history and family history were the same as on the previous admission. The physical examination was essentially the same, except for the abdominal examination. There was a scar over the right rectus muscle as a result of the previous operation. His right side was rigid and spastic all over. There was a palpable mass about the size of a small grapefruit which seemed to be somewhere in the center, between the right iliac fossa and the right costal margin. This mass was extremely tender to the touch. The left side of the abdomen was soft and tympanitic throughout and tenderness was not noted. A provisional diagnosis of malignancy of the colon with a secondary inflammation was made. The patient had a slight elevation of temperature; the pulse rate was not increased. An x-ray report two days after admission showed the following: The tone of the stomach was good; peristalsis was normal; the outline was regular, and the duodenum did not show any filling defect but appeared to be fixed. Examination five hours after the ingestion of a barium meal in the stomach showed the head of the meal in the sigmoid flexure. The entire colon was well filled. The cecum was irregular and showed a definite filling defect. Examination twenty-four hours after the barium meal showed

that the entire colon was practically empty, except for a small amount of barium in the distal portion of the colon. The region of the gallbladder did not show any evidence of stones.

The nonfilling, the irregularity and the hypermotility of the colon suggested a lesion in the cecum, which I believed would be revealed after x-ray examination made after a barium enema.

The patient's temperature gradually rose and the pulse rate increased. Pain was becoming worse and the mass was growing larger. Operation was advised on August 25, ten days after admission. The results of the urinalysis were negative, and the white blood count was 13,150. The Wassermann reaction of the blood was negative.

Operation—August 25: A right rectus incision, 4 inches (10.16 cm.) long, was made over the mass under ether anesthesia. On opening the peritoneal cavity, a large amount of a thick, greenish-yellow, foul smelling, pus escaped. Exploration revealed a large abscess cavity between the ascending colon and the lateral abdominal wall. There were many pockets which were freed so that one large abscess cavity resulted. The wound was closed in layers with two cigaret wicks and one rubber tube drain. On account of the severe inflammatory reaction and the large amount of pus in the abscess cavity, nothing further was done. Swabs from the abscess at the time of operation showed colon bacilli and a few short chained streptococcus.

The patient made an uneventful convalescence from the immediate effects of the operation. A considerable amount of pus drained until the sixth day when he developed a fecal fistula. On September 8, his head blocks were removed and he was taken off the Fowler's position; the temperature and pulse were normal. Fecal matter was still draining. On September 12, there was a marked inflammatory reaction around the edges of the wound. The temperature and pulse rate were normal. On September 20, the wound stopped draining. There was a sudden rise in temperature and pain was felt in the right side. Flaxseed poultices were applied and the wound began to drain again, but from that time on the patient became progressively worse and died on October 14, seven weeks after operation.

Postmortem Examination.—A large abscess was found in the right upper quadrant of the abdomen. The lowest portion of the right lobe of the liver formed the roof of the abscess cavity; the hepatic flexure, the medial wall, and the abdominal wall, the lateral wall. That portion of the liver which formed the roof of the abscess was gangrenous and necrotic. On the ascending colon near the hepatic flexure, there was a large perforation.

Pathologic Report.—Oct. 14, 1927: The specimen consisted of 5.5 cm. of terminal ileum, cecum, ascending colon with a portion of transverse colon, the whole measuring 30 cm. in length. The external surface of the cecum and the ascending colon was covered by a mass of densely adherent mesentery and scar tissue. At the hepatic flexure, this mass of adhesions was separated to form the wall of a pocket, roughly 7 cm. in diameter. A loop of jejunum was bound to the outer surface of the pocket by dense scar tissue. The wall of the pocket was made up of fibrous tissue 3 mm. thick, lined by a greenish, necrotic membrane. Beyond the pocket, the scar tissue stopped abruptly. A portion of omentum was adherent to the transverse colon. The adhesions were readily broken.

When the intestine was laid open, nothing remarkable was found in the ileum and jejunum. The cecum was lined by a reddish thickened mucosa. The wall of the ascending colon was thickened and fibrous and showed an irregular opening

3 cm. thick, representing a fecal fistula. The mucosa was thick, almost gangrenous about the opening.

On the ascending colon, distal to the perforation and under the site of the pocket was a shallow ulcer 1.5 by 1 cm. The base of this ulcer rested on surrounding fatty and scar tissue. Four other smaller ulcers were present, 1 cm.,

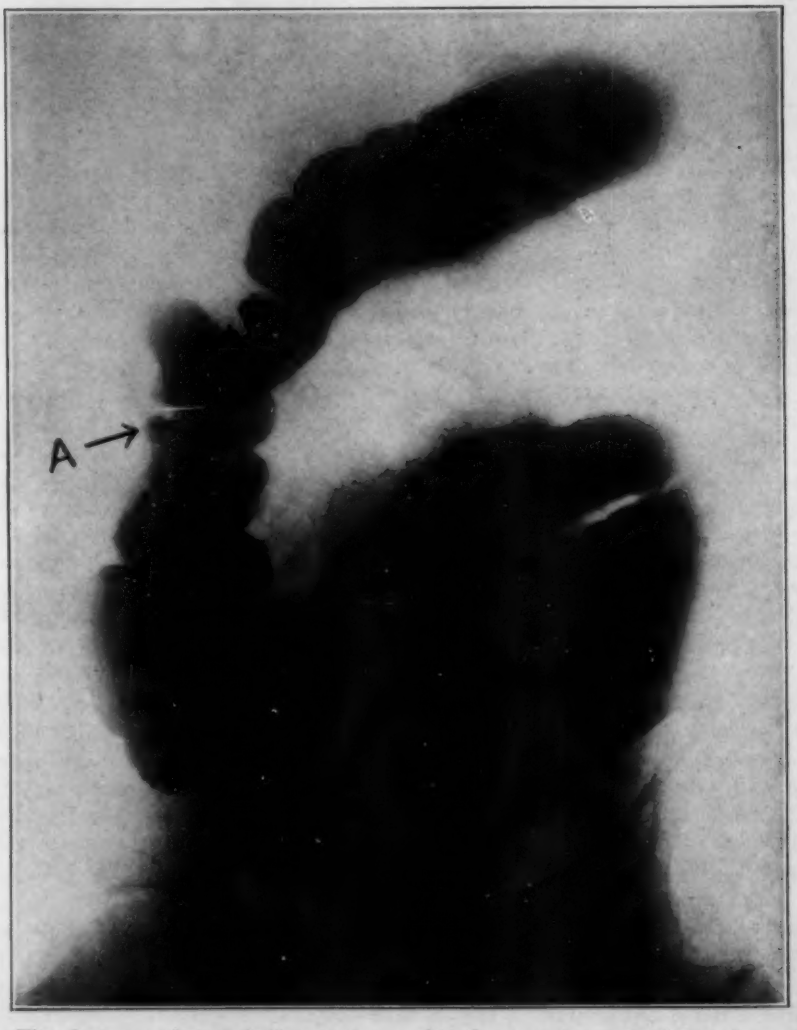

Fig. 2 (case 1).—*A* indicates the perforation on the ascending colon; the barium can be seen escaping in a small amount. The roentgenogram was taken following a barium enema.

4 cm. and 1 cm. in diameter, occurring at intervals of about 1.5 cm. The ulcers were pale, the edges flush with the surrounding mucosa. No communication could be found between the base of the ulcer and the surrounding scar tissue.

Microscopic examination of the ulcers showed the base made up of fibroblastic tissue which extended almost to the surface. The amount of granulation tissue was

small. A cellular exudate of lymphoid, plasma and polymorphonuclear cells, relatively small in amount, was present. The surface was covered by detritus. There was a marked increase in connective tissue beneath the ulcer, associated with atrophy and disappearance of muscle fibers and extending into surrounding fatty tissue. Large collections of lymphoid and plasma cells were present and a few polymorphonuclears.

Following the patient's death, the x-ray films were again studied. From the last x-ray examination, it now seemed rather evident that there was a small perforation in the distal portion of the ascending colon through which a small amount of barium could be seen escaping from the lumen of the bowel. This was overlooked prior to the operation. However, had this complication been noted, I hardly think that anything else could have been done for this man in the presence of the severe inflammatory reaction and the collection of pus in the abscess cavity. It seemed to me that proper drainage of the abscess was sufficient treatment. The fact that the fecal fistula was established and that the temperature returned to normal, would seem as though the abscess cavity had been properly drained and that there were no fecal contents remaining within the abdominal cavity. It is possible that there was another abscess which did not drain sufficiently and which caused necrosis of the liver, which ultimately resulted in the patient's death.

CASE 2.—*History.*—Miss M. G., aged 23, entered the surgical service of the Beth Israel Hospital on Feb. 23, 1923, with a complaint of severe pain in right lower quadrant. The onset of the condition began two days previously. At first, pain was generalized, then it seemed to localize itself in right lower quadrant. She vomited several times during the day of entrance. There had never been any abdominal pain previously. There was no blood in the vomitus, and the patient had never noticed any blood in the stools. Cough or hemoptysis was not noted. The menstrual history and the results of the urinalysis were negative. The leukocytosis was 13,000. The family history and past history were negative.

Physical Examination.—The patient was a well developed and well nourished young woman, lying in bed complaining of severe pain in right lower quadrant. The temperature was 99 F., the pulse rate, 90. There was nothing else of note except for the abdominal examination which revealed marked tenderness in the right lower quadrant with spasm of the muscles over this region. A definite tumor or mass could not be felt. Maximum tenderness was elicited over McBurney's point. Other than this, the abdomen was soft and tympanitic. A provisional diagnosis of acute appendicitis was made, and immediate operation was advised.

Operation.—The operation was performed under ether anesthesia on the night of entrance to the hospital. A right rectus incision was made. The appendix appeared normal. On the anterior lateral surface of the cecum, there could be seen and felt an indurated, inflamed, hard mass over which some omentum adhered. The omentum was separated from the cecum, and a piece covering this mass was excised, revealing what apparently was the base of an ulcer about the size of a quarter. There was no evidence of perforation. The ulcer was round and hard, resembling a callous peptic ulcer. Three mattress sutures of silk were placed through the serosa about the ulcer and it was covered with omentum. There was no evidence of tuberculosis as far as could be seen. A simple cyst of the right ovary about the size of an orange, together with the right tube, was also excised. The abdomen was then closed in layers in the usual manner. The patient made an uneventful convalescence and was discharged well in fourteen days.

Pathologic Report.—A small piece of omental tissue the size of a lima bean was examined. Gross examination showed that it was hemorrhagic, fat tissue. The tube was congested, thickened and showed fibrous adhesions. The cyst was the size of an orange, containing clear contents. The vermiform appendix, 6 cm.

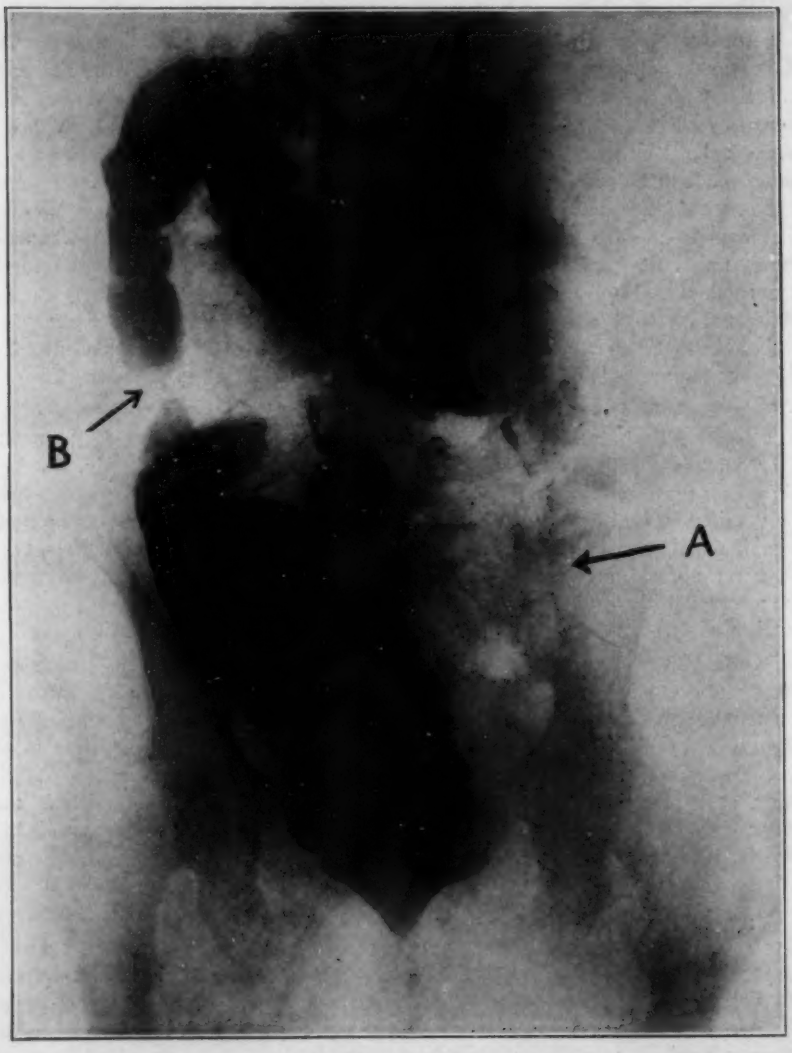

Fig. 3 (case 2).—*A* indicates the cecum, showing the filling defect three weeks after operation. *B* shows the spasm of the descending colon opposite the site of the lesion. This observation has been stressed by Carmen and others. (Roentgen-ray examination five years later showed a normal cecum.)

in length, did not show any acute signs of inflammation. Microscopic examination of omentum revealed some hemorrhage into the adipose tissue, with a marked polymorphonuclear leukocyte infiltration.

X-Ray Examination.—X-ray examination made three days before the patient was discharged from the hospital showed that the stomach was orthotonic with normal peristalsis; the outlines of the greater and lesser curvatures were regular; the duodenum was displaced toward the lesser curvature. Examination made three hours after a barium meal showed the head of the meal in the small bowel. The stomach was empty. Examination made six hours after the meal showed the head of the meal in the terminal ileum. This marked delay of barium entering the cecum was probably due to adhesions. Twenty-four hours after a barium meal, examination showed the height of the meal in the ampulla. The cecum and the ascending colon still contained barium. A barium enema was given. The cecum was irregularly filled. The condition at the ileocecal region was pathologic and was probably due to adhesions.

At my request, the patient returned almost five years after the operation for reexamination. She said that she was feeling well since operation. She did not feel any pain. Her appetite was good and bowel movements occurred daily without the aid of cathartics. The report of the x-ray examination was as follows: "The roentgen examination of Miss M. G., was made on December 10, 1927. Part radiographed:—Colon. The findings are as follows:—The examination of the colon with opaque enema shows the entire colon well filled, regular in contour, but rather atonic. The cecum is well filled. No abnormalities could be noted."

Case 3.—*History.*—Mrs. S. B., aged 32, married, entered the surgical service of the Beth Israel Hospital on March 26, 1922, complaining of bleeding from the rectum. Seven weeks before entrance, she noticed the bleeding after a bowel movement. The stool was rather large and bright red. There was a slight pain in the left lower quadrant which came on just before defecation. This pain radiated downward to the anal orifice. Since then, there had been three attacks similar to the previous one, with a considerable loss of blood each time. Diarrhea did not occur at any time, nor was there any mucus in the stools. The patient was rather constipated. Her appetite was good and she was not troubled with nausea or vomiting. Dysuria or hematuria were not noted. The family history was unimportant. The past history showed that she had had gonorrhea four years ago, but had not had any operations. Her husband was living and well; there was one child, also living and well. There had not been any miscarriages.

Physical Examination.—The patient was a well developed and well nourished young woman, lying in bed complaining of general weakness and pain in left lower quadrant. The skin was clear and of good color. There was nothing else of note except for the abdominal examination which revealed a soft and tympanitic region, although no mass or organs were felt. There was slight tenderness and resistance in the left lower quadrant.

Course.—On March 28, the patient had two loose, bloody stools. Considerable pain was felt in the left lower quadrant. On April 3, she developed an acute athritis of the right ankle. On April 6, she had a profuse hemorrhage from the rectum. She immediately went into collapse and became extremely pale; the mucous membranes were blanched, and she complained of air hunger. The pulse rate was 150 and barely perceptible. The blood pressure had fallen to 60 systolic, 35 diastolic. A blood transfusion of 750 cc. of blood was given. Following the transfusion, the pulse rate came down to 120 and the blood pressure rose to 90 systolic and 60 diastolic. Three hours after the transfusion, the pulse rate was still 120; the blood pressure had increased to 100 systolic and 50 diastolic.

On April 7, the patient was still bleeding from the rectum in small amounts. Sigmoidoscopy revealed a large, round, circumscribed ulcer about the size of a

half dollar on the right lateral and posterior wall of the rectum about 6 inches from the anus. In the center of the ulcer a bleeding point could be seen from which this profuse hemorrhage apparently came. The ulcer was packed with iodoform gauze, and the patient returned to bed in good condition. On April 9, the iodoform gauze was removed from the rectum. The patient never showed any signs of bleeding from the rectum since and was discharged well on June 12.

The results of the examination of the stool were negative. The patient was kept in the hospital until this time on account of the acute arthritis of the right ankle, which eventually disappeared. The results of the urinalysis were always negative. On May 25, the white blood count was 8,600; the red blood count, 4,252,000; polymorphonuclear neutrophils, 68 per cent, lymphocytes, 22 per cent, and large mononuclears, 9 per cent.

On March 28, 1928, the patient reported by letter that she had never had any diarrhea or bleeding from the rectum since she left the hospital. She has enjoyed perfect health and has since given birth to another child.

On April 16, 1928, at my request, the patient returned for a roentgen-ray examination of the colon. The following report was made: "The roentgen examination of Mrs. S. B., was made on April 16, 1928. Part examined: Colon (enema). The observations are as follows: The examination with an opaque enema shows the entire colon well filled and regular in contour with normal haustration throughout. No abnormalities could be noted."

TABLE 2.—*Age and Sex Incidence*

Ages	Number of Females	Number of Males
15–20	2	None
20–30	4	4
30–40	None	4
40–50	3	8
50–60	1	7
60–70	1	5
70–80	2	2

No age specified, 5; no age or sex specified, 2

AGE AND SEX

In this series of fifty-three cases, there were thirty-four males and seventeen females; in two cases the sex was not specified. One is immediately impressed with the fact that there are twice as many males as females. Apparently this seems to be a disease in which the male is more prone to be stricken than the female. The same observation has been made by many investigators regarding peptic ulcer.

The youngest female was 18 years of age, and the oldest 80; the youngest male was 20, and the oldest, 72. The average age for the group was 41.25 per cent years; for males, it was 43.5 per cent years, and for females, 41.06 per cent years, making the average age of both sexes about the same. Among the males, there was no age specified in three cases, and among the females, no age specified in two cases. Among the males, the greatest incidence occurred between the ages of 40 and 50; then 50 and 60, and then 60 and 70. Among the females,

the greatest number occurred between the ages of 20 and 30, then between 40 and 50. It would seem from this study, that the average age is the same for males and females and that it is not essentially a disease of the old.

ETIOLOGY AND PATHOGENESIS

Many observers are of the opinion that simple ulcers may occur in any part of the gastro-intestinal tract, and aside from ulcers of the stomach and duodenum, there are many cases reported of ulcers of the esophagus, the jejunum, the ileum and of the various segments of the colon. Bolton,[6] in his book on "Ulcer of the Stomach," said:

> It is not uncommon to find ulcers in other portions of the alimentary canal in association with gastric ulcer. Such an association is commoner in the case of ulcers of obviously infective origin, but also sometimes occurs in the simple, acute gastric ulcer. Such acute ulcers are found in the duodenum, esophagus, lower end of ileum and the colon.

In his series of twenty-four cases of ulcer of the stomach, additional ulcers in the same patient were found as follows: in the duodenum and stomach, fifteen cases; in the esophagus and stomach, four cases; in the ileum and stomach, three cases; in the stomach, two cases. In the present series which was studied, there was found four cases of ulcer of the colon in association with ulcer of the stomach (cases 4, 7, 46 and a case reported by Chvostek quoted by Quenu and Duval [4]), and two cases of ulcer of the colon in association with ulcer of the ileum (cases 2 and 12). Combes [7] has collected a series of thirty-six cases of simple ulcer occurring on the small and large bowel, divided as follows: seven in the jejunum, twelve in the ileum, five in the cecum, six in the colon, three in the sigmoid and three in the rectum. Gandy [8] studied a series of fifteen cases, divided as follows: end of the duodenum, one; jejunum, two; ileum, seven; cecum, two; hepatic flexure, one, and sigmoid, two. Vidal,[9] writing on "Ulcers of the Rectum," states: "Simple ulcers may be found throughout the whole length of the large intestine and even in the small intestine and that he has seen a few cases of chronic ulcers of the rectum." When the ulcer was single, he has only found it posteriorly. One of his patients, a man, aged 40, had symptoms of a simple ulcer of the stomach, and he believed that the ulcer of the rectum was of the same nature.

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6. Bolton, C.: Ulcer of the Stomach, London, Edward Arnold, 1913, p. 35-36.
7. Combes, C.: Simple Ulcer of the Intestine, Thesis, Toulouse, 1897.
8. Gandy, C.: Hemorrhagic Necrosis of Toxemia and Simple Ulcer, Thesis, Paris, 1899.
9. Vidal, E.: Rectum in "Dictionnaire encyclopedique des sciences medicales," 1874-1881, vol. 2, pp. 678-690.

Cruvielhier,[3] in his "Atlas of Pathologic Anatomy," expresses the opinion that the extremity of the intestine may be the seat of a simple ulcer similar to that of the stomach and duodenum.

It would seem that simple ulcer of the colon must be a manifestation of the same general disease as peptic ulcer, if there is a common etiologic factor in the production of these lesions on the various segments of the gastro-intestinal tract. This fact is particularly impressive since ulcer of the intestine is found not infrequently in association with ulcer of the stomach.

It is not unusual to find multiple ulcers of the duodenum and stomach in the same patient; there are numerous cases recorded that prove this. Recently Pickhardt [10] reported a case in which there were several ulcers in the stomach and duodenum, two of which, one in the stomach and one in the duodenum, had perforated. The fact that one is ready to accept the etiology as being the same when the lesion occurs both in the stomach and duodenum, is sufficient reason to accept the same etiologic factor, even though the lesion is farther down the intestinal tract. If one may correlate ulcer of the stomach with ulcer on any other part of the intestinal tract as having a common etiology, it is obvious that there may be a healed ulcer of the stomach and an active ulcer on the colon, or, from the same cause, a simple ulcer of the colon may exist alone. If ulcer of the colon and peptic ulcer are to be considered as having common etiology, the various theories that have been advanced for peptic ulcer must be taken up.

The theories that have been advanced regarding the initial cause of peptic ulcer may be classified according to whether or not the initial lesion is regarded as inflammatory, neurogenic, circulatory, bacterial, toxic, digestive or mechanical. The symptoms and the pathologic histology would seem to indicate that practically all of these theories might apply to ulcer of the colon. There are certain theories, however, that seem to explain the etiology more than the others.

The outstanding symptom seems to be constipation, of which there are twenty-one cases; constipation was not present in five cases, and in twenty-seven cases, it was not recorded. One must take into consideration that some of the early case reports were rather incomplete, and it is possible that in the twenty-seven cases in which constipation was not mentioned that there might have been many in which constipation was present. What rôle then can constipation play in the etiology of this condition? This would necessarily bring it under the head of the mechanical theory, either direct or indirect. This view is held by many observers (Quenu and Duval,[4] Soupault,[5] Delafield and Prudden [11]). Under this

10. Pickhardt, O. C.: Ann. Surg. **87**:143, 1928.

11. Delafield and Prudden: Textbook of Pathology, ed. 12, New York, William Wood & Company, 1923, p. 775.

theory, there could be produced the so-called stercoral or decubitus ulcer (case 49). "This type ulcer results in cases of obstipation from a crack or fissure in the mucosa which becomes infected and by extension develops into an ulceration" (Rankin [12]). According to Lyon and Bartle,[13] stercoral or decubital ulcers are mostly confined to elderly persons, and frequently to bedridden patients. "Through lack of the proper nourishment of the body, the coils of the intestines are not changed in their position sufficiently to aid the weakened bowel musculature to pass on its contents completely enough and stagnation occurs. Hardened masses of feces form in the colon and become agglutinated to the mucosa and when they are eventually pushed along through purging, or what not, ulceration quickly follows through secondary infection, attacking the denuded bowel wall. Perforation may take place." [13] This may explain certain ulcers in the rectum or in the sigmoid where the fecal mass may become exceedingly hard, but this can hardly be true in the cecum and ascending colon where the contents are normally fluid, and the patients in this series were not really old, with the possible exception of case 49.

Erosions, fissures and cracks might be caused by foreign bodies such as fruit pits and seeds (case 1). In this case, there was an obstruction due to an ulcer and the foreign bodies may have been lodged at the constriction later, making the occlusion complete.

Stengel and Fox [14] describe ulceration of the cecum in the following manner: "Typhilitis or cecitis may be due to irritation of the intestinal contents in consequence of constipation (stercoral typhilitis)." Typhilitis is probably generally of the simple catarrhal variety, but in obstinate constipation or obstruction of the colon, ulceration may occur. Perforations or extension to the surrounding tissues is the rarest of all consequences. Usually the latter is secondary to the inflammation of the vermiform appendix. Catarrhal inflammation of the cecum, according to Delafield and Prudden,[11] is not uncommon. It is usually produced by an habitual accumulation of feces in this part of the intestine. The course of the inflammation is usually chronic but marked by acute exacerbations. At first, the mucous membrane undergoes ordinary changes of chronic catarrhal inflammation. To this may succeed a slow suppurative inflammation which extends through the wall of the intestine and gives rise to ulcers and perforations. Through these perforations,

12. Rankin: Surgery of Colon, New York, D. Appleton & Company, 1926, p. 104.

13. Lyon and Bartle: Tice Practice of Medicine, Hagerstown, Md., W. F. Prior Company, 1924, vol. 7, p. 615.

14. Stengel and Fox: Textbook of Pathology, ed. 6, Philadelphia, W. B. Saunders Company, 1915, p. 636.

the feces may pass into the peritoneal cavity; or the perforations may be partly closed by adhesions, and abscesses or sinuses into the surrounding soft parts may be formed.

The question arises as to whether a catarrhal inflammation of the cecum or colon is primary, and ulceration a secondary manifestation. A controversy has existed for many years in regard to chronic gastritis associated with gastric ulcer. It has been held by some authors that ulcer was secondary to a chronic gastritis. I feel that it is the popular belief that a chronic gastritis is associated with, and probably secondary to, the ulcer. It would seem logical that the same condition exists in the cecum or in other parts of the colon. An ulcer being present, it may follow that secondary inflammation, either of a mild or severe character, may ensue.

According to Kocher,[15] the distention as a result of constipation, obstruction or impaction of feces produces a venous stasis at first in the corresponding segment of the mesenteric artery; ecchymosis follows, then there is a desquamation of the poorly nourished epithelium, and a superficial necrosis of the mucosa is produced. As a result of the experiments of von Greyerz and von Schimodeira,[16] there is a diminution in the arterial blood flow soon after distention occurs. The intestine becomes pale, and ulcers may appear even at this stage. After the distention is released, the intestine becomes deep red; a sign of venous stasis. As experimental ulcers occur as early as four or five days following distention with air, they feel that through venous stasis as a result of distention, nutritional disturbances occur in the intestinal wall, producing ulcers. Kocher and Prutz[17] consider venous stasis and its resulting thrombus formation the essential cause for the formation of these ulcers. According to Rosenheim,[18] the successive stages of ulceration are produced as follows: Intestinal stasis, progressive accumulation and more or less hardening of the contents, then fermentation; constant increase in the number and virulence of the bacteria, chronic inflammatory process, disorders of nutrition and ulcerating lesions. These are the theories of constipation as a mechanical factor: erosion by direct trauma, such as fecaloma, fruit seeds, etc., on the one hand, and the other, distention, due to overloading of the bowel, atony, or obstruction due to impacted feces.

The mechanical theory alone hardly seems to explain the etiology of this lesion, with constipation so universal and widespread. It would seem as though ulcer would be a much more common condition. It cer-

15. Kocher: Deutsche Ztschr. f. Chir., 1877, vol. 5.
16. Von Greyerz and von Schimodeira: Beitr. z. Chir. **22**:229, 1911.
17. Kocher and Prutz: Arch. f. klin. Chir., 1900, vol. 60.
18. Rosenheim: Ztschr. f. klin. Med. **54**:475, 1904.

tainly would not be considered that constipation, directly or indirectly, could cause simple ulcer of the small intestine. The same holds true of foreign bodies such as fruit pits, seeds, etc. Frequently, one sees patients who have swallowed foreign bodies which either pass through the gastro-intestinal tract or are removed surgically; the formation of an ulcer, as a result, is rare. It would seem that the foreign bodies are immedately enveloped by the soft fecal mass, and unless the object is a large one, its hard or rough edges seldom come in contact with the wall of the bowel. The theory advanced that venous stasis is a result of distention due to constipation could not explain the etiology in all cases. It does not seem that the etiology of simple ulcer of the colon can be any more explained than the etiology of peptic ulcer, and since I feel that simple ulcer is a manifestation of a general pathologic condition as gastric ulcer is, venous stasis as a result of a general condition such as a toxemia, regardless of its origin, may play an important rôle in the etiology, but not as a result of distention due to constipation.

In an exhaustive study of ulcerations of the gastro-intestinal tract, Gandy [8] came to the conclusion that simple ulcer of any part of the digestive tract is a manifestation of a general toxemia either of infectious or other origin. The toxemia produces an acute hemorrhagic necrosis by way of the blood stream due to hemorrhagic infarct; then follows sloughing, hemorrhagic erosion, ulceration of the mucosa, and finally, a deep ulceration. Rosenow strongly advances the bacterial theory based on the specific affinity of certain organisms for certain parts of the digestive tract. Rosenow, according to Mayo,[19] maintains that certain forms of bacteria injected through the blood stream cause ulcer of the bowel, especially of the cecum. Other forms cause ulceration in the alkaline small intestine or in the acid stomach. Dickinson [2] believes the infection takes place in the adenoid tissue of the cecum first and then extends to the mucosa giving rise to ulceration. Intestinal parasites may be an etiologic factor as described in case 48. Stahr [20] studied several cases in which he felt that the parasites were the causative factors in the production of simple ulcer of both the large and small bowel.

Under the circulatory theory, one must consider diseases of the vessel walls, such as atheroma and endarteritis obliterans. The idea that ulcer depends on diseased conditions of the blood vessel is supported by the observations in a few cases. Cases 21, 24 and 41 showed these vascular lesions. Cases 21 and 24 showed arteritis in the vicinity of the ulcer and the vessel filled with coagulated blood. Case 41 showed a perivascular

19. Mayo, C. H.: Discussion of paper by G. K. Dickinson, J. A. M. A. **78:** 1792 (June 10).

20. Stahr, H.: Intestinal Tumors in Children Caused by Trichocephalus, Deutsche med. Wchnschr. **48:**1274 (Sept.) 1922.

sclerosis, but no endarteritis. The capillaries were dilated and there was a diffuse hemorrhagic infiltration. Arteriosclerosis can produce infarcts and thrombi, lesions effecting a small portion of the colon or a large segment of the colon by cutting off the blood supply. Quenu and Duval [4] feel that the thrombosing endarteritis existed in the region of the ulcer in case 26. Lyon and Bartle [13] describe a thrombotic and embolic ulceration. They say: "Any part of the gastro-intestinal tract may be involved by this type of lesion, but it is seen more usually in the jejunum, ileum and colon."

Boss [21] holds that ulcers occur, as a rule, not as a primary lesion, but as a secondary manifestation, due to embolism or thrombosis of the arteries of the mesentery.

PATHOLOGY

Simple ulcer of the colon may be situated on any segment of the large intestine as shown in table 3.

The lesion may occur as a single ulcer or may be multiple. In thirty-seven cases, the ulcer occurred as a single lesion, and in sixteen cases

TABLE 3.—*Locations of Ulcer of the Colon*

Cecum	15	Splenic flexure	5
Ascending colon	12	Descending colon	2
Ascending colon and cecum	1	Sigmoid	11
Hepatic flexure	3	Rectum	4
Transverse colon	0		

they were multiple. When the ulcers were multiple, they were usually close to each other and there were never more than six. The lesion occurred most often on the cecum. The right half of the large bowel seemed to be a more frequent seat than the left, occurring in the right in thirty-one cases and on the left in twenty-two cases. This seems rather hard to explain since, as I have stated elsewhere, the contents of the cecum are normally fluid.[12] Since there were eleven cases with ulceration, either single or multiple, on the sigmoid, the question naturally arises as to whether these lesions were not diverticulitis or diverticulosis as the latter condition occurs so frequently on this portion of the colon. As these cases were all studied at postmortem examination or at operation, it seems that this question could be well ruled out. In a study of 118 patients who had diverticulitis, Judd and Pollock [22] found that there was no demonstrable narrowing of the lumen of the colon below the site of the diverticula, and furthermore, it is rather unusual to find diverticula in a colon which is markedly obstructed. It is interesting to note

21. Boss, W.: Perforated Ulcer of the Large Intestine, Beitr. z. klin. Chir. **140**:40, 1927.

22. Judd and Pollock: Ann. Surg. **80**:425, 1924.

that in the group of cases studied for ulcer the lesion tended to cause obstruction by a healed or healing stenosing cicatrix, while in diverticulitis there was no obstruction. In this group of patients obstruction was noted in ten cases. Hanseman [23] noted that false diverticula usually occur and are in close relationship to the blood vessels entering and leaving the intestine along the mesenteric border. This was not the case in this study; the ulcers usually occurred on the free border or anterior surface of the bowel, with the exception of case 17. A brief résumé of the gross lesion as studied by postmortem examination or at operation of ulcers of the sigmoid follows:

Case 5.—Postmortem examination showed that the proximal part of the colon was tremendously distended, while the rectum was much smaller. On the lower portion of the sigmoid there was a hard, annular stricture. It was so narrow that a small pin could hardly pass through it. The intestine above the stricture was covered with feces. The stricture was formed by an ulcer, the size of a 5 franc piece. The base was narrow and the edges raw, thickened and hypertrophied. Cancer could not be detected. The patient died of intestinal obstruction.

Case 8.—Postmortem examination showed six ulcers of the sigmoid. The ulcers were oval, resembling button holes and were all about the same size, and had the appearance as if the intestine was punched out. A certain number were perforated.

Case 14.—Postmortem examination showed an oval perforation, 5 cm. wide and 1 cm. long, on the middle part of the sigmoid. The edges were sharp and sclerotic. The mucosa had disappeared around the perforation.

Case 15.—Postmortem examination showed a perforating ulcer with sloping edges in the lowest portion of the sigmoid flexure. It opened into the abdominal cavity. There was a second ulcer beside it, which was ready to perforate. Its border was grayish.

Case 24.—Postmortem examination showed a linear tear on the sigmoid about 2 cm. long and surrounded by a small layer of fibrin. On opening the colon, ulcerations were found on the mucosa. One of these ulcers was deep, almost annular, and had perpendicular edges. The linear perforation already described was found to be at the base of this ulcer.

Case 25.—Postmortem examination was not made in this case, as the patient was cured following operation, and the lesion was not visualized, although the diagnosis of perforating ulcer of the sigmoid was made (see case reports).

Case 36.—Autopsy was not performed. Operation revealed a perforation the size of a 50 centime piece on the anterior surface of the sigmoid. The edges were slightly indurated for a short distance, and beyond the induration, the mucosa of the colon was apparently normal. The edges of the perforation were resected, and the loss of substance was sutured in two layers. Although the peritoneum and the intestine did not appear to be inflamed, extensive drainage was instituted on account of a large amount of feces in the abdomen. The patient made a good recovery from this operation.

Case 39.—Postmortem examination showed a small, round hole in the sigmoid from about 3 to 5 cm. in diameter, and about 12 cm. above Douglas' cul-

23. Hanseman, D.: Arch. f. path. Anat. **144**:400, 1896.

desac. The edges were infiltrated and its appearance was that of a perforated ulcer of the stomach. The stomach, small and large intestines were opened. The mucosa of the entire digestive tract was normal.

CASE 45.—Autopsy was not performed. Operation revealed fecal material in the abdomen and a perforation of the sigmoid. The walls of the colon were described as rotten, and they only tore on attempts to suture. The author was unable to get a clear idea of the abdominal condition, but thinks the perforation probably resulted from thrombosis. Evidence in favor of stercoral or foreign body was scanty.

CASE 49.—Postmortem examination showed an ulcer in the transition of the rectum into the sigmoid flexure of the colon. Rupture was due to a so-called decubitus ulcer. The perforation orifice was found to contain a hard fecal mass.

CASE 50.—In the lower portion of the sigmoid there was a perforating ulcer the size of a bean, from which feces were discharged. The tissue in the vicinity of the ulcer was infiltrated, but not reddened. The ulcer was closed by five longitudinal catgut sutures; two rows were made. Sigmoidoscopy four weeks after operation showed the mucosa normal for 22 cm. Analysis of the foregoing eleven cases would rather tend to prove that the lesions were not due to diverticulitis.

Diverticulitis of the right half of the colon is rather a rare condition. In a study of twenty-seven cases, Giffen [24] found diverticulitis situated on the various segments of the colon as follows: Hepatic flexure, one; transverse colon, one; sigmoid, twenty-two; rectum, two, and anal ring one.

The ulcer may be the size of a pea or the size of a dollar. There are all variations in sizes between the two mentioned. They may be annular, or their long axis may be parallel with the long axis of the intestine. The description of the lesion in the various case reports differ. In some cases the ulcer is described as oval, round, irregular with sharply cut edges, circular, or they may appear to be punched out. The mucosa around the ulcer grossly appears to be normal and when the ulcers are multiple the mucous membrane between them is also normal, but, occasionally, there may be a secondary inflammation of the mucosa. There may be a chronic, diffuse, hypertrophy of the intestinal wall. The ulcer may be hard, indurated and resemble gastric ulcer. The base has been described as being yellow, gray or white. The depth of the ulcer is variable; sometimes it is shallow, involving the mucosa and submucosa alone or the muscle layer or the serosa may form the base of the ulcer. Often there is a marked overgrowth of connective and inflammatory tissue which, in the process of healing, contracts and may form an annular or longitudinal constriction, producing either partial or complete obstruction. Rarely, there is hemorrhage from the ulcer. Severe

24. Giffen: The Diagnosis of Diverticulitis of the Large Bowel, J. A. M. A. **59**:864 (Sept. 14) 1912.

hemorrhage has occurred, but more often when the lesion is in the rectum, due to erosion of a blood vessel (cases 26, 47, 49 and my case 3).

In all cases, the initial lesion is acute, and an acute ulcer results. This ulcer may heal rapidly, but, on the other hand, it may extend, while a secondary inflammatory thickening occurs, corresponding in degree to the chronicity of the ulcer. A chronic ulcer, which is produced in this way, may likewise continue extending, so that a description of the ulcerative process comprises not only that of the formation of the initial lesion, but also that of the extension of acute and chronic ulcer.

However, the ulcer may continue as a chronic simple latent ulcer, giving rise to mild abdominal symptoms or, as stated before, may heal. The opposite course is perforation. From a study of this series of cases, it would seem that simple ulcer of the colon was rather prone to perforate; however, it is impossible to make this a definite statement since, in the majority of the cases studied, the diagnosis was only made when perforation occurred and it is likely that the simple latent ulcers go unrecognized. In this study of fifty-three cases, forty-two ulcers perforated and eleven did not perforate. The perforation is generally in the center of the ulcer which may be small or large. The omentum or a loop of adjacent bowel may tend to wall off and form a pocket, as in my case, one with an abscess resulting; this condition occurred in twelve cases; or the contents of the colon may be expelled into the general peritoneal cavity, giving rise to general peritonitis. The localized abscess resulting from a perforation often can be felt as a distinct tumor through the abdominal wall. The pus from this abscess may burrow and form sinuses into the liver, kidneys or any adjacent organ (cases 16, 17 and 18).

Vascular lesions about the ulcer, such as nonobliterative and obliterative endarteritis, thrombosis; complete obliteration by cicatricial fibrous tissue, penetrating into some of the capillaries have been described (cases 21, 24 and 26). There is an absence of epithelium and mucosa, and in some cases, of submucosa and muscularis. The layers lying underneath the destroyed ones have also lost their normal structure and given place to a cicatricial tissue, poor in cells but rather vascular. Ecot and Richard [25] found a direct infiltration of polymorphonuclear leukocytes and large phagocytic cells. There was also perivascular sclerosis.

Mintz [26] describes the serosa as considerably thickened with newly formed dilated vessels and a small round cell infiltration. He could not

25. Ecot, F. C., and Richard, A.: Perforation of a Simple Ulcer of the Ascending Colon: Microscopical Examination, Bull. et mém. Soc. anat. de Paris **92**:138, 1922.

26. Mintz, W.: Simple Ulcer of the Colon, Zentrabl. f. Chir. **50**:1208, 1923.

demonstrate thrombosing endarteritis. It is interesting to quote the histologic observations in my case 1. The base was made up of fibroblastic tissue; the amount of granulation tissue was small. There was a cellular exudate of lymphoid, plasma and polymorphonuclear leukocytes, relatively small in amount. The surface was covered by detritus. There was a marked increase in connective tissue beneath the ulcer, associated with atrophy and disappearance of muscle fiber and extending into surrounding fatty tissue.

SYMPTOMATOLOGY

The symptoms of simple ulcer of the colon must be divided according to whether or not the lesion is a chronic ulcer or whether perforation has taken place. Again, the symptoms of chronic latent ulcer may be subdivided according to whether the ulcer is a simple, shallow ulcer or, as a result of healing, cicatrization may occur, forming either a constricting band or a pseudoneoplasm due to an overgrowth of connective and inflammatory tissue, protruding within the lumen of the bowel causing more or less obstruction.

A simple, shallow ulcer may give rise to no symptoms or to vague, indefinite abdominal pain, which is often attributed to constipation. This lesion is difficult to recognize and is usually found during an exploratory operation. The constricting or occluding ulcer is associated with pain, which at times may be acute and usually does not bear any relation to meals. This type of ulcer gives rise to constipation due to partial or complete obstruction. There were ten cases in this group, or almost 19 per cent, with definite intestinal obstruction (cases 1, 10, 11, 12, 13, 28, 29, 34, 36 and 44).

Constipation as an associated condition with ulcer of the colon seems to be stressed strongly by the European writers. It is obvious that constipation must occur where more or less obstruction exists. Also, any localized, pathologic lesion on the colon might give rise to a spastic condition due to motor disturbances which, in turn, could produce constipation. In this series, marked constipation was present in twenty-one cases; in five cases it was not present, and in twenty-seven cases it was not mentioned. As I have mentioned before, constipation may have been much more prevalent. Melena is occasionally present (case 13). Severe hemorrhage is rare, but does occur, as in cases 47 and 49, particularly when the ulcer is located in the rectum (case 26 and my case 3).

The symptoms of perforated ulcer present an entirely different picture and may also be subdivided as follows: 1. When the ulcer perforates and there is a spontaneous discharge of the intestinal contents into the free abdominal cavity, a sudden, severe, sharp pain occurs at the point of perforation. The abdomen at first is scaphoid and retracted;

shock is present, then to be followed by the usual signs and symptoms of general peritonitis as arises following perforation of any hollow viscus. 2. Ulcer of the colon sometimes forms an intra-abdominal tumor, owing to a small perforation which causes an extensive infiltration of the abdominal wall. There is a surrounding peritoneal reaction; adhesions of the small intestine or omentum form a true tumor which can be often felt. This type usually gives rise to localized pain and rise in temperature; if it is on the right side, like appendical abscess. There were twelve cases, about 22.5 per cent, in which a definite, palpable tumor could be felt (cases 19, 21, 24, 25, 27, 28, 29, 31, 39, 40, 48 and 51, and in cases 40 and 48, the ulcer had caused an intussusception. Occasionally, these symptoms subside due to a local, natural, defense reaction. The virulence of the invading organisms is lowered. The perforation is closed over by a layer of fibrin, then layer on layer of inflammatory tissue is deposited about the localized perforation. The temperature may become normal and even the pain may disappear, and the patient believes himself to be well. The inflammatory tumor persists however, and can be easily palpated. Essentially, the symptoms of perforated ulcer simulate those of peritonitis, either local or general.

DIAGNOSIS

The diagnosis of ulcer of the colon is difficult to establish before perforation has taken place. Simple ulcer of the colon has no pathognomonic signs by which the diagnosis can be made. In not one of the cases in the series studied was the diagnosis made before perforation or operation took place, with the exception of the case in which the ulcer was located in the rectum. Constant, fixed pain, associated with constipation, should make one suspect ulcer as well as cancer in the differential diagnosis. At best, this lesion can only be suspected, and, with the aid of the x-ray, the diagnosis may be made. The sigmoidoscope and proctoscope will be of considerable assistance when the ulcer is in the rectum, or low down in the sigmoid. X-ray examination will help to rule out a diagnosis of colitis and cancer.

When the ulcer has perforated and signs of peritonitis, either local or general, have made themselves evident, the diagnosis is usually made at operation. If an abdominal tumor can be felt, together with acute inflammatory signs, perforation of an ulcer should immediately be considered. The first impression is a perforated appendix, acute cholecystitis, volvulus, strangulation or intussusception, but whatever diagnosis is made, on account of the gravity of the symptoms, operation is usually performed at once. It is then that a knowledge of ulcers of the colon appears to be particularly valuable. In fact, in a peritonitis involving the right iliac fossa, the finding of a normal appendix should cause

the possibility of a perforation of the colon to be considered. The same perforation may be found to be the cause of localized peritonitis in other parts of the abdominal cavity. After a negative exploration of an organ, which is believed to be diseased, the surgeon should remember that a chronic ulcer or a perforation of the large intestine may exist in patients whose past history shows nothing with the exception of constipation, and he should make a careful examination of the walls of the colon.

It is of particular importance to differentiate, during an operation, between a pseudoneoplasm due to an ulcer, carcinoma and diverticulitis, since in cancer, the most radical treatment possible should be carried out.

TREATMENT

In order to discuss the rational treatment of patients with simple ulcer of the colon, chronic latent ulcer and perforated ulcer must be considered separately. Since this lesion has never been correctly diagnosed in the unperforated state prior to operation, it seems rather difficult to advise operation for the condition, even though a correct diagnosis has been rendered. In spite of the fact that only fifty-three cases have been reported in the literature, it seems that the lesion must be much more prevalent and goes unrecognized. The number of perforations that have been reported is relatively small in number, which makes me believe that the ulcer or ulceration must heal itself spontaneously rather often. A certain number of ulcers do perforate, and therefore operation, before this accident occurs, is rather a simple procedure and may be a life saving measure if the ulcer be a perforating one. Apparently, simple suture of the serosa over the base of a shallow ulcer is sufficient treatment, as proved in case 40 and in my case 2. When the ulcer has caused definite constriction, due to a healed contracting scar, a simple plastic operation may be sufficient rather than extensive resection. If, however, the ulcer, as a result of inflammation, has produced a pseudoneoplasm protruding within the lumen of the bowel causing definite obstruction, the usual resection of that portion of the bowel must be performed. The surgical treatment for persons with simple ulcer in the unperforated state may be divided as follows: (1) for simple shallow ulcer without constriction or obstruction, simply bringing the serosa over the base of the ulcer as reinforcement is sufficient; (2) for the constricting band due to a perfectly healed ulcer, a plastic operation is sufficient; (3) for an obstructing type of ulcer with secondary inflammation, the treatment must be resection.

The treatment in cases of perforated ulcer must be immediate operation. If the perforation is immediately visualized and opens directly into the peritoneal cavity, the ideal method would be to suture the perforation

without the excision of the edges, cleansing the abdominal cavity of fecal and purulent material and establishing adequate drainage. Certainly, resection would be contraindicated in the presence of a more or less generalized peritonitis. If the perforation has been walled off and a localized abscess has developed, incision, evacuation of the abscess, suture of the perforation whenever possible, without too much manipulation, and adequate drainage would be sufficient, rather than performing a more complete and dangerous operation.

When the ulcer is sufficiently low in the rectum, cauterization and tamponade is probably enough, as in case 26. In my case 3, tamponade was enough to check the hemorrhage, and the ulcer apparently healed itself.

SUMMARY

Fifty-three cases of simple, nonspecific ulcer of the colon are reported. Fifty cases were collected from the literature and three were personal cases. There were thirty-four autopsies, one incomplete autopsy and two deaths without autopsies in this series. Fifteen patients were cured as a result of operation; two of these patients had intestinal resections performed and the resected colon was studied grossly and microscopically. The others were studied only by visualization at time of operation. In one case, it was not stated as to whether the patient survived or not.

Simple ulcer, analogous to gastric or duodenal ulcer, may occur on any part of the alimentary canal from the esophagus to the rectum. Ulcer of the colon is a distinct and definite pathologic lesion which probably occurs more frequently than is suspected; the diagnosis usually being made only when the ulcer has perforated, or during an exploratory laparotomy or at the time of a postmortem examination.

Simple ulcer of the colon is usually the cause of the so-called inflammatory tumor or pseudoneoplasm and as a result of which, intestinal obstruction is produced either by the inflammatory tumor or constriction, usually due to a healing ulcer or the cicatrix of a healed ulcer.

The etiologic factor is probably not constipation and therefore stercoral or decubital ulcers are not common. Simple ulcer of the colon is not infrequently associated with gastric or duodenal ulcer. The fact that ulcer occurring on the stomach and on the first portion of the small intestine, namely, the duodenum, is accepted as having the same etiology, it is logical to believe that ulcer of the stomach occurring with an ulcer on another segment of the gastro-intestinal tract, namely, the colon, have the same etiology. Therefore, it seems that ulcer of the colon is probably a secondary and local manifestation of a general pathologic condition as gastric ulcer, and that the same etiologic factor may be the cause of simple ulcer on any segment of the alimentary canal.

There were approximately twice as many cases of ulcer of the colon in males as in females. This is not a disease of old age; the average proving that it occurs before middle life.

Perforations occurred in forty-two of the fifty-three cases reported.

REPORTS OF CASES FROM THE LITERATURE

CASE 1 (Marchesseaux,[27] 1837).—*Perforation of Ulcer of the Rectum.*

A woman, aged 45, an alcoholic with marked constipation, died of acute peritonitis. At autopsy, a stricture was found in the upper part of the rectum. Above the stricture, many grape seeds and fruit pits were found. The stricture barely permitted the passing of one's little finger. There was a large circular ulcer on its surface about 1 inch (2.5 cm.) wide; the edges were formed by slightly swollen, pale mucosa. The base was pale and was formed by the submucous connective tissue. In the center, a small perforation was noted. The intestinal coats were only slightly thickened. There was no evidence of ulcer in any other point of the gastro-intestinal tract. All the other viscera were normal.

CASE 2 (Rogee,[28] 1838).—*Chronic Ulcers of the Intestine Without Tubercular Perforation.*

A woman, aged 61, with a delicate constitution, for three years had been having frequent attacks of colic accompanied by nausea and vomiting. Diarrhea had not occurred. The condition gradually disappeared. She had no primary acute infection. She suddenly had violent pains in the abdomen and died with signs of peritonitis. At autopsy, the lungs showed evidence of old healed tuberculous process in both apexes. The viscera were normal. The peritoneum showed signs of acute peritonitis. There were six narrow ulcers in the last fifth of the small intestine opposite the attachment of the mesentery. The ulcers were elongated and their great diameter was perpendicular to the axis of the intestine. The base of some of the ulcers was formed by the peritoneum only; in others the base was formed by the muscle layer, and in some the submucous connective tissue formed the base. Granulations were not seen on the surface of the ulcers. Only one ulcer in the colon, situated high up in the rectum, perforated. It presented the same characteristics as the ulcers of the small intestine, but it was round and about the size of a 2 franc piece. The mucosa of the gastro-intestinal tract was normal with the exception of the ulcers. The mesenteric glands were normal and there was no evidence of tuberculosis present.

CASE 3 (Greenhow,[29] 1848).—*Perforating Ulcer of the Splenic Flexure.*

A man, aged 72, had an attack of renal colic six months before consulting a physician. Examination showed a calculus in the bladder. Lithotomy was performed on November 7. On November 9, slight abdominal pain, rapid pulse and marked constipation developed. Mild mercuric chloride was given on November 10; the patient grew weaker and had diarrhea, death occurred that morning. At autopsy, the peritoneum showed signs of peritonitis. The coats of the bladder were markedly thickened. The prostate was indurated and large. A small calculus

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27. Marchesseaux: Stricture of the Upper Part of the Rectum with Old Ulceration, Bull. Soc. anat. de Paris **12**:343 (Dec.) 1837.

28. Rogee: Chronic Ulcers of the Intestine Without Tuberculosis; Perforation; Acute Peritonitis; Death, Bull. Soc. anat. de Paris **13**:181 (Aug.) 1838.

29. Greenhow: Perforating Ulcer of the Splenic Flexure, Path. Soc. Newcastle and Calestead, December, 1848.

was found in the bladder. There was a large amount of feces in the left hypochondrium. There was a small perforation at the splenic flexure from which the intestinal contents escaped. The colon presented ulcers at a few other points. These other ulcers had almost penetrated through the entire thickness of the intestinal coats. Immediate cause of death was peritonitis due to perforation.

CASE 4 (Lebert,[30] 1861).—*Perforating Ulcer of the Colon.*

A man, aged 47, who had always been in good health, had sharp pains in the abdomen fifteen days before admission to the hospital. Pain was localized along the course of the colon. He became markedly distended, and later constipated. There was nausea and vomiting. On admission to the hospital, he had signs of peritonitis and died two days afterward. At autopsy, there was general peritonitis. The abdomen was filled with gas and there was a large amount of feces in the right iliac fossa. The lesser curvature of the stomach presented an ulcer about the size of a 5 centime piece, about 10 cm. from the cardia. The small intestine was normal. There was a rounded perforation a little larger than a 5 centime piece at the splenic flexure surrounded by an area of injection and false membranes. The ulcer when seen from the mucous surface showed the edges to be almost perpendicular.

CASE 5 (Idem).—*Ulcer of the Sigmoid; Intestinal Obstruction; Death.*

A man, aged 47, had always been in good general health, although he always complained of marked constipation and hemorrhoids. He continued to work until ten days before his death. At this time he began to vomit and became markedly constipated. In spite of all that could be done, the constipation persisted and his abdomen became more distended, but without pain. He was much nauseated and vomited continually until his death.

At autopsy, the abdomen was found to be markedly distended. There were only a few adhesions. The only changes were found at the lowest portion of the sigmoid. Above this point, the colon was tremendously distended while the rectum was much smaller. At this point, there was a hard annular constriction. The stricture was so narrow that a small pin could hardly be passed through. The intestine above the stricture was filled with feces. The stricture was formed by an ulcer the size of a 5 franc piece. The base was yellowish and the edges broad, thickened and hypertrophied. After careful examination with a microscope, cancer could not be detected. There was a single simple ulcer which had caused the obstruction of the intestine, by increasing hypertrophy of the intestinal walls.

CASE 6 (Dujardine,[31] 1864).—*Perforating Ulcer of the Splenic Flexure.*

A physician, who became suddenly ill with pain on April 12, described the pain like that of a sharp blow which caused him to sit down and rest. Shortly after, he was able to walk home complaining of a pain in the left hypochondrium. His physician made a diagnosis of colitis and applied leeches which seemed to give him relief. At the end of twenty days, he was advised to go to the country for a rest. He did not improve and complained of indigestion and pain. On returning, he consulted Prof. Orsey on June 5, who made a diagnosis of tumor of the spleen or adjacent organs and advised waiting until the next day because he wished to see the color of the patient's skin before he made his final diagnosis. He had several attacks of pain with diarrhea and fever and died on June 24. At autopsy,

30. Lebert: Perforating Ulcer of the Colon, Traité d'anatomie et pathologie, générale et spéciale, 1861, vol. 2, p. 206.

31. Dujardine: Perforating Ulcer of the Splenic Flexure, Soc. d sc. méd. et naturelles de Bruxelles, September 5, 1864.

a perforation of an ulcer of the sigmoid colon was found. This ulcer communicated with a closed pocket of cellular tissue as a result of the inflammation so that the fecal contents had not escaped into the free abdominal cavity and this explains the length of his illness.

CASE 7 (Claus,[32] 1856).—*Perforating Ulcer of the Descending Colon.*

A man, aged 47, had had typhoid twenty-five years previously and pneumonia fifteen years previously. On May 8, 1855, he had pneumonia again. On the seventeenth day of his illness, dyspnea increased and he had violent pains in his abdomen. He had diarrhea, followed by constipation which could not be relieved. He died two days after entering the hospital with evidence of peritonitis. At autopsy, on the lesser curvature of the stomach, an ulcer the size of a 5 centime piece was found and there were several other small ulcerations on the greater curvature. The small intestine was slightly hyperemic and contained four or five round worms. There was a rounded perforation the size of a 5 centime piece on the posterior wall of the descending colon and the edges were perpendicular. Nothing else was found in the gastro-intestinal tract. The lungs showed evidence of pneumonia.

CASE 8 (Dickinson,[33] 1867).—The patient was a woman, aged 42, who died in St. George's Hospital on Aug. 26, 1863. She said that she had had constipation and a bilious fever several months before admission. About a month before her death, she had pains in the left iliac fossa and died with symptoms of a circumscribed peritonitis. At autopsy, a cavity the size of a cocoanut was found in the left iliac fossa. This cavity was partly empty. The anterior wall of the cavity was formed by the abdominal wall and the posterior wall was formed by intestinal loops which were adherent to each other by means of solid lymphoid tissue. The cavity contained pus and feces, and communicated with the sigmoid by means of a certain number of perforating ulcers. About half a dozen ulcers were seen on opening the colon. The ulcers were oval, resembling button holes and were all about the same size. They were close together, parallel to each other and perpendicular to the long axis of the intestine. They did not present any thickening, and they had the appearance as if the intestine was punched out. The muscular layer was destroyed. There was no evidence of tuberculosis.

CASE 9 (Idem).—*Perforating Ulcers of the Descending Colon.*

The patient was a man, aged 42, who died at St. George's Hospital on Feb. 10, 1865. He had attacks of pain in the left hypochondrium for several years. These attacks have become worse recently. Pain was associated with constipation and he would be relieved by cathartics. The patient became edematous; his urine contained albumin and he had diarrhea which could not be controlled. This appeared to be the cause of his death. At autopsy, a large cavity filled with black feces was found. The cavity was made up of the diaphragm, spleen and descending colon. There were two small narrow ulcers in the upper part of the descending colon which opened into this cavity. There was no thickening of the intestinal coats. There was no other lesion present with the exception of the ulcers. These ulcers were in the form of button holes, close together, and parallel and their long diameter was perpendicular to the long axis of the intestine. No evidence of tuberculosis was found.

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32. Claus, A.: Spontaneous Perforation of the Colon, Inaug. Diss., Zurich, 1856.

33. Dickinson: Perforating Ulcer of the Transverse Colon, Tr. Path. Soc. Lond. **18**:101, 1867.

CASE 10 (Robin,[34] 1869).—*Ulcer of the Descending Colon.*

A man, aged 66, entered the hospital on May 19, 1868. He had had gout since he was 19 years of age. On the morning of May 20, he had a severe attack of gout and there followed severe constipation for five days with slight abdominal distention. He was given a purgative, which caused one bowel movement. On May 26, the patient demanded his discharge from the hospital. This was refused on account of the increase in the abdominal distention. The constipation and distention increased. His condition became serious on May 29. There had not been any stools for several days. He now had a weak pulse, coldness of the extremities and vomiting. Galvanism was applied to the abdomen without results. It was impossible to pass an esophageal sound to the upper limit of the rectum. The patient's condition became much worse and death followed. At autopsy, the intestine protruded on opening the abdomen. The colon was distended with gas and liquid feces; its walls tore at the slightest traction. There was no evidence of peritonitis or strangulation. There was an ulcer the size of a 5 franc piece in the middle portion of the descending colon. The ulcer was of recent date. Nothing was found which could explain the arrest of the sound in the rectum. Crystals of sodium urate were found in the articulations of the toes.

CASE 11 (Costin,[35] 1870).—*Perforating Ulcer of the Ascending Colon.*

A man, aged 45, had had abdominal pain, constipation and slight fever. Palpation showed that the colon was filled with hard feces. Various purgatives such as castor oil, mild mercuric chloride, senna and croton oil were administered without any notable result. The patient died twelve days after he was first seen. At autopsy, the small intestines were found to be distended. There was evidence of peritonitis. There was a certain amount of exudate in the peritoneal cavity and it had a fetid odor. The right side of the omentum was gangrenous and presented small disseminated abscesses. There was an old ulcer with sharp borders on the posterior surface of the middle third of the ascending colon. It had perforated through the entire thickness of the intestinal wall. The other viscera were normal.

CASE 12 (Lepine,[36] 1870).—*Annular Ulcer of the Intestine; Perforation of the Small Intestine; Stricture of the Large Intestine, Autopsy.*

A woman, aged 70, died in the service of Dr. Charcot shortly after admission to the hospital. There was no clinical information. The abdomen was large, and the peritoneum was markedly injected. Several loops of intestine were covered with false membranes. The small intestine and the ascending and transverse colon were dilated, but the descending colon was of normal size. There was a stricture at the splenic flexure. The little finger could not be passed through this stricture. The intestinal walls were greatly thickened through the whole extent of the stricture. There was a round ulceration of small size a few millimeters above the stricture. The mucosa of the large intestine was normal throughout with the exception of the small ulcer. In the terminal ileum, there were six round ulcers penetrating to the muscle layer. The ulcer which was most distant from the ileocecal valve was 2 or 3 cm. in diameter, and the borders were thin. Careful examination showed that this perforation had not been artificially produced during autopsy and that it produced the terminal peritonitis. There was no evidence of

34. Robin: Ulcer of the Descending Colon, Thèse Denaire, Paris, 1869.

35. Costin, J. G.: Perforating Ulcer of the Ascending Colon, Brit. M. J. **2:** 247, 1870.

36. Lepine: Perforation of the Small Intestine; Stricture of the Large Intestine, Bull. Soc. anat. de Paris **45:**27 (Jan.) 1870.

any tuberculous lesions. The mucosa stopped abruptly where the ulcers commenced. The objective appearance was that of a simple ulcer. The stricture at the splenic flexure was due to the cicatrix of a healed ulcer.

CASE 13 (Chouppe,[37] 1870).—*Simple Ulcer and Stricture of the Large Intestine.*

A man, aged 32, had colic almost continuously for two months. He vomited after every meal, and colic returned after vomiting. The vomitus and feces were black. The patient was very constipated and would go for seven or eight days without a bowel movement; this condition would be followed by diarrhea. His appetite was good. At autopsy, there was no evidence of tuberculosis. There were signs of peritonitis in the abdominal cavity. The cecum and ascending colon were enormously distended. A stricture was situated at the hepatic flexure. Palpation at this area did not show any indurated tissue. After having incised the large intestine above and through the stricture, a large ulcerated area with irregular borders was found in center of stricture. No evidence of malignancy or any other lesion was found in the gastro-intestinal tract.

CASE 14 (Demange,[38] 1879).—*Old Perforation of the Sigmoid Flexure, Obstructed by Epiploic Adhesions; Rupture of These Adhesions; Acute Peritonitis.*

A man, aged 72, had sharp abdominal pains one evening, and was found dead the next morning. At autopsy, generalized acute peritonitis was found. There was an oval perforation 5 mm. wide and 1 cm. long on the middle part of the sigmoid flexure. The edges were sharp and sclerosed. The mucosa had disappeared around the perforation. The base of the ulcer was occupied by epiploic masses which were adherent to the edges and which undoubtedly formed a stopper up to the time of rupture.

CASE 15 (Mickle,[39] 1883).—*Double Perforating Ulcer of the Sigmoid Loop of the Colon.*

A man, aged 45, had an extremely severe pain in the right iliac region near the median line after having taken a purgative. There were signs of peritonitis due to perforation. He died twenty-four and a half hours after this sudden onset. Autopsy revealed generalized peritonitis. There was a perforating ulcer with sloping edges in the lower portion of the sigmoid loop. It opened into the abdominal cavity. There was a second ulcer beside it which was ready to perforate. Its borders were grayish. There was stenosis of the mitral valve and biliary calculi, but no calculi were found in the intestine or peritoneum.

CASE 16 (Bradbury,[40] 1884).—*Ulcer of the Cecum, Complicated with Abscesses of the Liver.*

A man, aged 30, was admitted to the hospital on May 28, 1884, with a condition, the first symptoms of which appeared on April 25 and consisted of abdominal pain on the left side, chills, vomiting, diarrhea, fever and loss of weight. On admission, there was dulness over the base of the left lung. The abdomen was soft but painful in the left iliac fossa. Cough was noted. Examination did not show the presence of a rash. There was an alternation of the chills and fever. The

37. Chouppe: Simple Ulcer and Stricture of the Large Intestine, Bull. Soc. anat. de Paris **45**:322 (May) 1870.

38. Demange, E.: Old Perforation of the Sigmoid; Acute Peritonitis, Rev. méd. de l'est, August, 1897, p. 500.

39. Mickle: Double Perforating Ulcer of the Sigmoid, Brit. M. J. **1**:637, 1883.

40. Bradbury: Ulcer of the Cecum, Complicated with Abscess of the Liver, Lancet **2**:637, 1884.

abdomen became distended and painful on June 25, and the area of dulness around the liver increased. Acute pain developed in the right flank on July 9, and there was a large pleural effusion of the right side. The patient died on July 16. Autopsy revealed the right pleura filled with yellowish pus which had a nauseating odor. There was a fistula communicating between the pleura and an abscess of the liver. The liver contained many abscesses of various sizes, the largest of which was about the size of a lemon. This was the abscess which had ruptured through the diaphragm. There was an ulcer the size of a pea in the intestine near the attachment of the appendix to the cecum. It was surrounded by congested mucosa, but there was no perforation of the wall. There was no evident cause of the ulcer; the appendix was normal. The cecum was fixed in the right iliac fossa by old adhesions. The hemorrhoidal veins and the inferior mesenteric veins were filled with clots and pus. There were no lesions in the rectum or small intestine.

Case 17 (Oliver,[41] 1895).—*Acute Perforating Ulcer of the Ascending Colon.*

At autopsy, there were two longitudinal ulcers in the colon 2½ inches (6.27 cm.) above the ileocecal valve. The larger ulcer was about 1 inch (2.5 cm.) in diameter, while the other scarcely admitted the tip of a goose feather. The surrounding tissues appeared to be normal as well as the tissue between the two ulcers. The two perforations were of rounded form and their edges were perfectly regular. The different tunics of the intestine had undergone necrosis to an equal degree; however, the muscle layer was retracted so that there resulted a prolapse of the mucosa which appeared to be redundant.

The larger opening, seen from the outside, presented the appearance of an artificia anus while the smaller opening could scarcely be discovered because of the retraction and the elasticity of the intestinal tissue.

The ulcers were situated exactly in the middle of the posterior surface of the ascending colon. The posterior surface, which was not covered by peritoneum, was lined by the connective tissue of the fascia which filled the lumbar space and the anterior surface of the right kidney. The necrosis had gradually extended through the intestinal wall, forming a tunnel through which fecal material escaped into the areolar tissue separating the colon and the right kidney from the subadjacent tissues. The peritoneum showed only slight signs of inflammation; only the lower edge of the liver presented traces of fibrinous exudate over a limited area.

Case 18 (Parker,[42] 1887).—*Ulcer of the Ascending Colon.*

A girl, aged 18, entered the hospital on Sept. 16, 1885 for perityphilitis; she had been treated with rest and poultices. She improved and left the hospital in six weeks. On Dec. 24, 1885, she noticed a swelling in the right thorax redness, tension and dyspnea. The tumor increased in size; she coughed and expectorated. She returned to the hospital. The abscess was punctured on January 12, but only a half ounce (14.7868 cc.) of pus was obtained. An incision was made on January 13. The cavity was irrigated, but there was little pus. The patient's general condition improved at first and then became worse; cough, profuse expectoration, night sweats and hectic fever developed. At about the middle of February it was believed that there were signs of subacute pulmonary tuberculosis.

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41. Oliver, J.: Acute Perforating Ulcer of the Ascending Colon, Lancet **1**: 424, 1885.

42. Parker, A.: Ulcer of the Ascending Colon, St. Barth. Hosp. Rep. **23**:213, 1887.

There was a continuous discharge of pus from the thoracic wound. The functions of the digestive tube were perfectly regular. The cachexia increased toward the end of May, and the patient died on June 2, 1886.

Autopsy showed that the head was normal, and the pericardium was distended with about 1 pint (473.2 cc.) of clear and limpid fluid. Pericarditis was not noted. The pleura were adherent and contained a cloudy liquid rich in fibrin. Examination of the lungs showed generalized bronchopneumonia with suppuration and formation of small disseminated abscesses. The left lung was in a worse condition than the right. Peritonitis was not found in the abdomen. The ascending colon was firmly united to the liver at the hepatic flexure. It formed one of the walls of the cavity filled with pus. This suppuration extended into the right lobe of the liver, which was softened and friable. It communicated with an opening in the right flank by means of a narrow fistula situated in the posterior axillary line between the ninth and tenth ribs. There was only a small amount of pus. The tenth rib was necrotic at the level of the sinus. On opening the ascending colon, a small ulcer the size of a pea was found. It was evidently of old formation and had perforated the wall of the intestine and opened into the area of suppuration. The ulcer was circular and had rounded edges. It did not present any trace of active evolution. There was nothing to indicate the origin of this ulcer. The rest of the digestive tract was normal. The spleen showed amyloid degeneration. The kidneys were fatty and slightly amyloid. The pelvic organs were normal.

Case 19 (Idem).—*Ulcer of the Colon and Cecum.*

A woman, aged 49, fell from a wagon on Aug. 14, 1887. She entered the hospital on August 16 for contusions of the face and the right thigh and abdominal pains. There was no history of previous abdominal disorders. Her general condition was grave. She was constipated for six days following the accident and had continual, greenish and aqueous vomiting. She had a profuse evacuation on August 20, undoubtedly due to a purgative; her stools were fetid and liquid; the vomiting ceased; she had fever; the diarrhea persisted. Examination of the urine did not show any albumin. On August 28, a diffused swelling was found at the hepatic flexure. The patient was weak and her temperature was normal. A tumor gradually developed and became of considerable size. It was situated at the hepatic flexure of the colon. There was a gradual weakness; the stools were irregular and were dark, fetid and liquid. On September 20, she had cough, pneumonic expectoration and fine crepitant râles at both bases. The diarrhea and cachexia continued, and the patient died on October 20. At autopsy, the heart and pericardium were found to be normal. The pleurae were also normal. The right lung showed hepatization of the base and disseminated suppuration. The left lung showed generalized bronchopneumonia and the bronchi were filled with pus. Chronic generalized peritonitis was found in the abdomen; the intestinal loops were adherent to each other. There was a collection of fecal matter and pus around the cecum and ascending colon. This collection extended upward as far as the diaphragm, passing in front of the liver. The pus and feces were contained in adhesions. There was a circular ulcer at the base of the cecum opposite its peritoneal insertion. This ulcer had rounded edges and had perforated through the intestine. It did not present any sign of recent evolution. It was the size of a 3 pence piece. A second similar ulcer, twice the size of that of the cecum, was found on the anterior wall of the ascending colon about 3 inches (7.6 cm.) from the cecum. There were no signs of typhoid, dysentery or tuberculosis. The intestines were soft and were in putrefaction. The right kidney showed congenital atrophy; it was the size of the kidney of an infant, while the left kidney was twice the normal size. The other abdominal viscera were normal.

CASE 20 (Ollier,[43] 1892).—*Slough of the Cecum; Perforation.*

A man, aged 56, suddenly had a violent abdominal pain. There were signs of peritonitis and he died on the seventh day. Autopsy showed generalized peritonitis. There were two perforations of from 1 to 3 mm. in diameter, on the anterior surface of the cecum 2 or 3 cm. from the ileocecal valve. They were on the internal surface of the intestine in the center of a gangrenous area which was the color of dead leaves and was 5 or 6 cm. long and 1 to 1.5 cm. wide and surrounded by an area of inflammation. The rest of the intestine was normal and the appendix was intact.

CASE 21 (Morel and Secheyron,[44] 1895). *Ulcer of the Cecum; Inflammatory Pseudoneoplasm; Iliocecal Resection; Cure.*

A man, aged 46, entered the hospital on Sept. 17, 1895, for digestive disorders and progressive general weakness. He was tall and thin. On admission to the hospital, his face had an earthy tint, and his features showed a condition of general weakness. The disease began on July 14, 1895. Suddenly and without any cause, while working, he had had a severe pain in the right iliac fossa. Nevertheless, that day and the following days he had been able to continue working, but he had had almost continual pain. This pain presented exacerbations at fixed periods, coming on two hours after each meal. In the interval, there was rather a sensation of weight or heaviness at the level of the cecum, than a true pain. The digestive functions continued to be regular; there was no constipation or diarrhea. Stools were passed every day without increasing the pains and without discharge of blood and melena. His appetite remained excellent, but in spite of these conditions, he lost weight and strength.

From the beginning of the disease, the patient had noticed a swelling in the right iliac fossa. This swelling was situated between the anterior superior spine of the ileum and the umbilicus. Palpation was not painful, and showed an ill defined, elongated, lobulated tumor which was mobile in all directions. The dulness was not absolute on percussion. There were no adhesions to the skin. The glands in the groin were normal. In the presence of this tumor, and especially on account of the patient's straw-yellow color and earthy tint, a diagnosis was made of cancer of the cecum. The tumor seemed to be well limited, nonadherent, and surgical operation seemed to be justified. The patient was placed on a milk diet for eight days and he was given benzonaphthol to produce intestinal asepsis as much as possible. Another careful examination was made before giving the anesthetic. A small tumor could be clearly felt at the upper part of the right iliac fossa. It was mobile and the size of a small apple. It seemed to be surrounded by a sort of resistant and lobulated tissue. After the patient was under the anesthetic, it was impossible to palpate this tumor and only a diffuse swelling could be felt.

Operation was performed. Palpation of the abdomen showed a tumor which protruded into the cecum. This tumor was surrounded by an indurated zone which extended along the external wall of the cecum to 6 or 8 cm. from the valve. There was an oval, thickened plaque, about the size of a 2 franc piece, situated on the adherent border of the mesocolon 10 or 12 cm. from the valve. It was milky white. The mesenteric glands near the cecum were large and hypertrophied. The appendix was normal. The diagnosis at operation was cancer of

43. Ollier: A Case of Double Perforation of the Cecum, Loire méd. **11**:3 (Jan. 15) 1892.

44. Morel and Secheyron: Ulcer of the Cecum: Pseudoinflammatory Neoplasm; Ileocecal Resection; Cure, Soc. de méd. de Toulouse, 1895.

the cecum. The postoperative course was uneventful. The patient became cured and left the hospital on the fortieth day. He was in perfect health two and one-half months later and had gained 12 Kg. in weight.

Examination of the cecum showed that the tissue removed at operation consisted of the cecum, appendix, ascending colon, a part of the transverse colon and 2 cm. of the small intestine. The total length was 35 cm. The intestine was opened following along the external border. This incision divided a loss of substance into two almost equal parts at the level of the cecum. It was an oval ulcer with sharp outlines and the edges were about 2 mm. high. The base was indurated, pale gray and of uniform coloration. This ulcer was almost opposite the valve of Bauhin; its small vertical axis measured about 3.5 cm.; its great axis was in a transverse direction and measured 7 cm.; and the two extremities of this transverse great axis were each 1.5 cm. from the commissures of the ileocecal valve. The mucosa of the cecum was not absolutely normal; it appeared to be slightly infiltrated, and in addition, it presented white streaks which radiated from the edges of the ulcer. These streaks were almost fibrous and they flattened the folds of the mucosa, which was pink and shining. The mucous membrane of the intestine was normal in all of the other portions. The ulceration was in the middle of a large indurated plaque of almost cartilaginous hardness. This induration was situated on the antero-external and posterior walls of the cecum for a distance of about 7 cm. Its edges decreased in hardness and gradually disappeared. At the level of the induration, the intestine was considerably thickened and measured more than 1 cm. The intestine seemed to be of normal thickness in all other parts.

Microscopic examination at low magnification showed that the ulcer had destroyed the entire thickness of the glandular layer, the musculature of the mucosa and the most superficial part of the mucous membrane. The glands were increased in length about 5 cm. The connective tissue was much more dense than that of normal intestine; it formed tight, thick bundles which stained intensely with acid fuschsin. The muscular layers were hypertrophied; the internal layer measured 3 mm. and the external layer 2 mm. The serosa was also slightly thickened. Under higher magnification, it was found that in the vicinity of the ulcer the glandular layer was infiltrated in its entire thickness by a large number of embryonal cells. At certain points, these cells formed masses resting on the musculature of the mucosa. The epithelial lining cells of the intestine and those of the tubular glands appeared to be normal. The base of the ulcer was formed by the serosa; it was made up of connective tissue bundles irregularly sectioned by the ulcerative process. In the superficial parts especially, it was infiltrated by a large number of embryonal cells and at certain points there were small interstitial hemorrhages. Vascular lesions could be seen below the ulceration; certain arterioles showed obliterating endarteritis; others were thrombosed and filled with coagulated blood; finally there were larger vessels which presented slight traces of endarteritis. In many places the connective tissue around these vessels was thicker and tighter than elsewhere. The vascular lesions were far from being generalized; most of the arterioles subadjacent to the ulcer appeared to be completely normal. Sections of the colon above the ulcer showed that the coats of the intestine were normal and there were no vascular changes.

Case 22 (Southam,[45] 1895).—*Perforating Ulcer of the Cecum.*

A man, aged 67, who had suffered from symptoms of incomplete obstruction of the intestine for several months, died suddenly a short time after admission to the

45. Southam: Perforating Ulcer of the Cecum, Brit. M. J. **1**:254, 1895.

hospital. At autopsy, a stercoral ulcer was found on the posterior wall of the cecum. There was a small perforation at the center. The appendix was normal but the cecum and colon were distended by feces. The distention was apparently due to atony of the intestines, because there was no obstruction along the course of the intestine.

CASE 23 (Guinard,[46] 1896).—*Perforating Ulcer of the Hepatic Flexure of the Colon.*

A man, aged 27, while in good health, suddenly was seized with violent abdominal pain. Laparotomy was performed thirty-six hours later. There was diffuse peritonitis. The appendix was simply congested, but was excised; its cavity was free. The patient died thirteen days after operation. At autopsy, there was a purulent area containing gas at the junction of the ascending and transverse colons. The omentum was thickened and black. There was generalized peritonitis at the hepatic flexure and a perforation the size of a 5 franc piece was found. The intestinal mucosa was greatly congested. No other ulcers were found in the digestive tract. The perforation of the colon was the primary cause of the symptoms.

CASE 24 (Morel and Rispail,[47] 1896).—*Ulcer of the Pelvic Colon; Perforation.*

The patient was a man, aged 67, who entered the hospital on April 26, 1896. His hereditary history was negative. He had never been seriously ill; however, since infancy, he frequently had dull pains in the abdomen, undoubtedly due to chronic constipation; his stools had never been regular and at times he had profuse evacuations of solid material. At the time of the first examination, constipation had lasted for one month. During this time, the patient had no desire to go to the stool. His appetite was preserved. Food did not produce any discomfort or sensation of weight; he had no fever and his facies were not altered.

Examination showed that the abdomen was large and tense. A tumor of cylindrical shape could easily be seen under the skin. It began in the cecal region, ascended obliquely and became lost in the left hypochondrium. Palpation showed that this swelling was hard, resistant and of uniform consistency throughout its whole extent. Percussion showed marked loss of resonance. The left side of the abdomen seemed to be effaced in contrast to the distention of the right side. On palpation, it seemed that the sigmoid and the descending colon were empty, and over these regions percussion gave the usual abdominal resonance.

Rectal examination did not show any tumor or stricture. The fingers seemed to penetrate into a sort of excavation, as if the rectal ampulla was dilated in all directions. It was completely empty and did not contain any fecal matter. On account of these different symptoms, a diagnosis was made of intestinal occlusion. Although the distended loop extended obliquely upward and to the left, and did not follow the course of the ascending and transverse colons, it was believed that the cause of the obstruction was at the splenic flexure. The nature of the obstacle could not be determined. Cancer was eliminated. The patient was placed on a milk diet and a purgative enema was given on the 22d. Castor oil was given on the 23d and repeated on the 24th; three stools. On the 25th, he had a spontaneous stool. There was pain at the level of the umbilicus, which soon radiated. He did not vomit. Death occurred suddenly on the 26th. The patient had refused any operation. At autopsy, the abdomen was greatly distended

46. Guinard: Perforating Ulcer of the Hepatic Flexure, Thesis Paris, June, 1896, no. 324.

47. Morel and Rispail: Ulcer of the Sigmoid; Perforation, Soc. de méd. de Toulouse, 1896.

and considerable gas escaped on opening the peritoneum. The true pelvis was filled with a cloudy, seropurulent fluid containing fibrinous false membranes. On examining the contents of the abdomen, one single loop of intestine was found to be of enormous size and was recognized to be a portion of the large intestine. It extended obliquely from the right iliac fossa to the left hypochondrium. On elevating it slightly, a linear tear about 2 cm. long and surrounded by a small layer of fibrin was found on the right lateral surface. Intestinal matter was escaping through this opening. Great difficulty was experienced in determining which portion of the large intestine was affected. However, it was evident that this distended loop was the sigmoid which had greatly increased in size and had left the true pelvis to extend into the abdomen in front of the viscera. After having extracted this loop from the abdomen, and after having sectioned it longitudinally, it was seen that it formed an enormous sac containing many kilograms of fecal material which was greenish brown, soft and did not contain scybala or foreign bodies. The intestinal walls were indurated and thick. Ulcerations were found on the mucosa. One of these ulcers was deep, almost annular and had perpendicular edges. The linear perforation already described was found at its base.

Microscopic examination, at low magnification, showed that the ulcer was funnel-shaped; the loss of substance occurred only in the mucosa and the musculature of the mucosa. The base of the ulcer was formed by the submucosa; the edges were perpendicular and were formed by the glandular layer of the mucosa and the musculature of the mucosa. One of the edges of the glandular layer was separated from the muscular layer which was destroyed, and at this level there was a free space separating this layer from the muscular layer. The other layers of the intestine were normal in appearance. Under higher magnification, it was found that the anatomic elements composing the glandular of the intestine in the vicinity of the edges of the loss of substance stained poorly; the nuclei of the glandular epithelial cells, as well as their protoplasm and the connective tissue of the wall of these glands had undergone coagulation necrosis. A short distance from the edges of the ulcer it was found that the anatomic elements preserved their normal structure. The base of the loss of substance was formed of loose connective tissue containing several round cells in its meshes; although the inflammatory process was relatively slight at this level, it was found, however, that this part of the submucosa contained many more embryonal cells than at other points of this tunic distant from the loss of substance. There was no appreciable ulceration of the vessels or the other layers of the intestine. In this case, the ulceration was chiefly due to necrosis of the superficial layers of the mucosa accompanied by a moderate inflammatory process in the subadjacent part of the mucosa.

CASE 25 (Gamgee,[48] 1898).—*Perforating Ulcer of the Sigmoid Flexure of the Colon.*

A man, aged 28, had ulcerative colitis in August, 1897. He suddenly had an acute pain in the inferior region of the abdomen on November 1. His temperature ranged between 100 and 101 F. for several days. A circumscribed tumor gradually developed in the left iliac fossa. The tumor was incised and about 3 drachms (11.09 cc.) of pus were obtained from a localized intraperitoneal abscess. Twelve days after operation, gas and the contents of an enema escaped through the wound. The fistula closed and the patient recovered.

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48. Gamgee: Perforating Ulcer of the Sigmoid, Brit. M. J. **1**:950, 1898.

CASE 26 (Quenu and Duval,[4] 1902).—*Simple Hemorrhagic Ulcer of the Rectal Ampulla.*

A woman, aged 50, entered the hospital for profuse rectal hemorrhages. The hereditary history was negative. She had three children, two of whom died of diarrhea and convulsions; the living child was 16½ years of age. There had also been two miscarriages. She had been treated for metritis after the first miscarriage, and had been sent to mineral springs. She had not had any other illnesses. The digestive tract functioned normally; digestion was good; she did not have diarrhea or constipation. The stools were normal and did not contain any mucus or membranes. She used alcohol somewhat to excess. The first rectal hemorrhage occurred in November, 1899. It appeared without any definite cause and was profuse. She had syncope, and her pulse became weak. A subcutaneous injection of salt solution became necessary. The second hemorrhage occurred in May, 1900; it was arrested by rest in bed and ice. She became weak. In July, 1900, there was a third hemorrage accompanied by syncope, which was controlled by ice and rest in bed. The fourth hemorrhage occurred in July, 1901; ice and rest in bed were instituted as before. The fifth hemorrhage occurred on May 25, 1902. This was perhaps the most severe hemorrhage, and rapidly caused syncope. Two injections of aqueous extract of ergot and an injection of salt solution were given. On the following day, the patient did not have hemorrhage. The urine did not contain sugar or albumin.

She entered the hospital on June 22. Rectal examination showed only some anal varicose veins. With the speculum 8 cm. above the anocutaneous line at the junction of the posterior wall with the right lateral wall, the following was found: (1) a superficial ulceration with a pink base, 5 or 6 cm. long, from which the hemorrhage occurred; (2) a cicatricial band situated just to the side of the ulcer which bled easily. The ulcer was cauterized with the thermocautery on June 22, and the hemorrhage ceased. Tampons were placed in the rectum. Menstruation commenced on the afternoon of June 23, and during the night there was a severe rectal hemorrhage which required salt solution. The uterine hemorrhage soon ceased. Tampons were placed in the rectum. The ulcer was on the road to cicatrization on June 27, and on July 12 healing was complete and the patient left the hospital.

Microscopic examination revealed an ulcer of the rectal ampulla with arterial thrombosis. Serial sections were made following the axis of the extirpated ulcer. On each side of the sections it was found that the rectal mucosa had an absolutely normal cylindrical epithelium. On the base of ulcer, the epithelium and the mucosa were lacking. In their place there was a depression 1 mm. deep and 1.5 mm. wide. The base of this depression was formed by a fibrous zone rich in migratory cells (especially phagocytes) and covered with many red cells. It also contained some smooth muscle fibers which seemed to belong to the muscularis of the intestine. This fibrous zone rested on a dense, sclerous, cicatricial tissue which contained few cells and was intensely stained by fibrous tissue stains. This tissue not only formed the deep portions of the ulceration, but it extended on each side below the mucosa which rested directly on it without the interposition of a muscularis mucosa or any of the other layers of the intestine. The mucosa was thinner than normal and its interstitial tissue contained many leukocytes. The cicatricial sclerotic tissue contained a large number of capillaries. In addition, it surrounded a certain number of small veins and arterioles. The largest of these arterioles presented a remarkable disposition. It was found that in all sections the lumen was completely obliterated by a cicatricial fibrous tissue containing

several capillaries, although the lumen was perfectly outlined by the elastic membrane and the middle tunic. The presence of cicatricial tissue containing vessels in the interior of an artery can only be interpreted as representing an old thrombosis of this vessel.

CASE 27 (Tedenat,[49] 1902).—*Enormous Retrocecal Abscess; Multiple, Large Perforations at the Origin of the Colon; Secondary Suture; Cure.*

A man, aged 40, who had had good health up to the age of 26 years, while living in Brazil for four years, had had syphilis, malaria and beriberi; he drank alcohol to excess. He returned to France in 1889. He had chronic constipation with abdominal pains, frequent cough and was only in fair general condition. The present disease began on April 29, 1901, with sudden pains in the entire right side of the abdomen, radiating toward the thorax and groin, without vomiting, and without complete constipation. He remained in bed for twenty days with thirst and fever. His abdomen was slightly distended and was painful and tender on the slightest pressure.

The patient entered the hospital on May 21. He was pale and thin. His temperature was 40.2 C. (104.3 F.), and his pulse rate was 104 and small. There was sharp pain in the entire right half of the distended abdomen. The skin over the right iliac fossa was red. The cecum was distended and gave a dull tympanitic sound. Cough was paroxysmal and there was induration of the right apex.

Operation was performed on May 22. Roux's incision for appendicitis was made. The abdominal wall was indurated and bled freely. As soon as the peritoneum was opened, about 500 cc. of thick fetid pus mixed with gas and fecal matter escaped. The cecum was greatly dilated and was reddish-violet. There were grayish, false membranes on its antero-external surface, which was adherent to the parietal peritoneum. The appendix was of almost normal appearance and was found underneath the false membranes. On passing the finger behind the cecum, a large glassful of pus escaped. There were two rounded perforations, 12 and 15 mm. in diameter on the posterior surface of the colon a little above its origin. These perforations were about 3 cm. apart. At 4 cm. above the highest perforation, there were three other rounded perforations in a row, from 7 to 8 mm. in diameter. Two large rubber drains were placed behind the cecum and the entire perforated portion was surrounded with iodoform gauze and a large cotton dressing was applied. The patient passed a good night and on the next morning was much better; the temperature was practically normal; the pulse rate 69, and weak. On June 26, the patient's general condition was good. The fecal fistula still discharged. It was decided to close the fistula.

An operation was performed under chloroform. An incision 26 cm. long was made parallel to the first incision at one finger's breadth from the parietal fistula. The cecum was reached and the colon was separated from the abdominal wall. The intestine was almost normal. There was practically no change in the perforations on the posterior surface of the colon. Each of the perforations was closed with a purse-string suture, and the suture line was covered with peritoneum. A large rubber and gauze drain was inserted to keep the cecum and colon away from the incision. Dressings were made with compresses moistened with salt solution. In the evening, his temperature was practically normal; his pulse rate 98, and strong.

The patient left the hospital on July 26 with a granulating wound about 6 cm. long and from 1 to 2 cm. wide. The fistula closed. The intestine was not distended. There was no pain. The patient had a good appetite; his bowel move-

49. Tedenat, quoted by Quenu and Duval.

ments were regular and his general condition good. Tuberculous granulations were not seen either around the perforations or on the peritoneum. There were no lesions resembling cecocolic tuberculosis of the hypertrophic form. Hence, tuberculosis may be eliminated as a cause of these rounded perforations with perpendicular edges. Can the lesions be attributed to syphilis? The syphilis had been well treated.

CASE 28 (Idem).—*Retrocecal Abscess in the Iliac Fossa and in the Fatty Tissue Around the Femoral Vessels in the Middle Part of the Thigh; Perforation of the Posterior Wall of the Cecum; Appendix Little or not Diseased; Death Due to Chronic Septicemia.*

A man, aged 52, of good health, had had typhoid at the age of 18 years. On Aug. 10, 1891, after eight days of complete constipation, his abdomen became painful and he began to vomit. His condition was serious for four days and was attributed to intestinal obstruction. An enema produced a profuse evacuation. The same painful symptoms recurred on September 8. The maximum pain was in the right iliac fossa with radiation to the thigh. He did not vomit. There was an abundant discharge of gas through the anus. The stools became normal at the end of a week and the abdomen returned to its normal size, but the pain persisted in the iliac fossa and right thigh. He had continuous fever. A physician was called on September 20, and found a deep swelling in the iliac fossa extending to the middle part of the antero-internal surface of the thigh. The swelling increased progressively, remaining deep in the abdomen and becoming more superficial at the internal surface of the thigh. The patient entered the hospital on Oct. 12, 1891, thirty-four days after the beginning of this second attack. Although he continued to take milk and bouillon without vomiting, he was pale, thin, feeble and had hectic fever. There was a deep induration in the right iliac fossa. It extended to the median line without any marked redness of the skin and continued downward to the middle part of the thigh, elevating the vessels and thrusting them inward. The skin was red and adherent over the swelling in the thigh and showed fluctuation. There was an enormous edema of the entire leg. The intestine was moderately distended, but the abdomen was only slightly painful on pressure, except in the iliac fossa.

On October 13, an oblique incision 8 cm. long was made a little outside of the resonant protrusion formed by the cecum. Several adhesions were broken at the infero-external part of the thickened cecum and about 1 liter of fetid pus escaped. There was a perforation about 2 cm. in diameter on the external part of the posterior surface of the cecum. The purulent cavity was prolonged downward toward the true pelvis. A second incision was made 2 cm. below the crural arch. It was 6 cm. long and was parallel to and outside the femoral vessels. A counter opening was made behind the middle part of the tendon of the fascia lata. The crural focus communicated with the iliac focus through the crural canal. The cecum was adherent to the anterior abdominal wall. The purulent cavity was subperitoneal. The appendix was searched for in vain. Four drains were inserted. There was great improvement of the general condition for about twelve days. The patient took 2 liters of milk, bouillon and wine.

On October 25, there was a profuse discharge containing fecal material. The pains increased and the fever rose. Two days later, an incision of 5 cm. within and parallel to the first was made in order to reach the internal surface of the cecum, for the purpose of searching for another abscess, resecting the ileocolic portion or making an anastomosis. Some thin adhesions were separated. The appendix was practically free and was of normal appearance. It was below and behind the cecum. It was rapidly resected. The operation had to be discontinued

because the patient was taking the anesthetic poorly. The incision was sutured, leaving the middle portion open for a large gauze drain. His condition remained the same for about eight days. The temperature oscillated around 39.5 C. (103.1 F.) and his pulse was between 115 and 130, but he had no vomiting. The patient died with hectic fever on Nov. 20, 1891, due to the large suppurating focus in the thigh.

Complete autopsy could not be made. During the course of the operation the following conditions were found: (1) a large perforation on the posterior surface of the cecum without any tuberculous nodules; (2) a subperitoneal iliac abscess with extension of the pus into the sheaths of the femoral vessels, and (3) integrity of the appendix on macroscopic and microscopic examination. There was a perforating ulcer, probably not due to tuberculosis. It could also be stated that it was not due to the appendix. It was impossible to state whether or not it was a mechanical ulceration.

CASE 29 (Idem).—*Large Stercoral Fistula Following Multiple, Simple Perforating Ulcers at the Origin of the Colon; Ileocolic Resection; Cure by Lateral Anastomosis.*

A man, aged 68, entered the hospital on Jan. 20, 1897. He was of robust constitution and he had not had any serious diseases. He used alcohol somewhat to excess. His general health was good, considering his age. He did not suffer from chronic constipation. He was a heavy eater. He had had two attacks similar to the one for which he entered the hospital. The first attack occurred ten months before with pains in the right iliac fossa, which soon radiated to the entire abdomen. There was mucous and bilious vomiting and absolute constipation without emission of gas. The abdomen was distended. The urine was scanty. These symptoms all disappeared in four days after a purgative.

The second attack occurred on Jan. 1, 1897, with sudden sharp pain in the right iliac fossa. Nausea was present with some vomiting. The patient became constipated. Three days later, an enema produced a profuse, fetid evacuation, with a considerable quantity of blood mixed with the feces. Hemorrhoids were not present. There was a feeling of relative well-being after these attacks. The appetite was satisfactory, and there was only slight constipation. There was vague tenderness at the upper part of the right iliac fossa. The third attack occurred on January 16, following a purgative which did not produce evacuation. There was colic and sharp pains in the right iliac fossa, which radiated throughout the abdomen.

The patient arrived at the hospital on January 20, after riding for three hours on the railroad train. His features were drawn; his eyes were hollow; his voice was husky, and his tongue was dry. The pulse was small and rapid. Bilious vomiting occurred at intervals, but was not painful; the abdomen was distended and somewhat painful throughout. Nothing was found in the rectum nor in the hernial regions. There was regular distention of the entire intestine including the sigmoid flexure. It was more marked at the lower part of the ascending colon. In that region there was an area where there was the greatest sensitiveness on pressure and this area was also the site of the most intense pains since the first attacks. There was slight induration of the wall with redness of the skin at this level.

An incision 5 cm. long was made vertically at this place. The entire thickness of the wall had not been incised when there escaped a large amount of black, fetid, fecal matter mixed with gas. There was an abscess undergoing a subacute course. The pains, nausea and vomiting disappeared rapidly. The gangrenous

débris was eliminated in seven or eight days, and a large intestinal fistula became established. The general condition of the patient improved rapidly, but the fistula still discharged abundantly.

On March 16, operation was performed for the cure of the artificial anus, under an anesthesia of a mixture of alcohol, chloroform and ether. A flap of skin 6 cm. long and 3 cm. wide was excised. There was a slight prolapse of the intestinal mucosa. It was separated with difficulty, inversed and sutured with a Lembert suture. The tissues were friable. The principal perforation was rounded and 2 cm. in diameter. Above this perforation there were two others, slightly smaller, separated by bridges of intestinal wall 1 to 2 cm. wide. These perforations were located at the origin of the colon. A resection of the ileocecal segment with partial resection of the colon was decided on. The lower end of the parietal incision was enlarged downward and inward and the upper end directly upward. The cecum was separated, drawn outside the wound with the terminal portion of the ileum and the first 10 cm. of the ascending colon. Forceps protected by rubber were applied on the colon 8 cm. from the ileocecal valve and on the ileum at the same distance from its opening into the cecum. A wide, lateral anastomosis was made between the ileum and colon over a distance of 5 cm. The terminal sections of both ends of the intestine were sutured. The edges of the wide and thick mesentery of the large intestine were sutured. All of these sutures were made rapidly with Lembert sutures of fine silk. The intestinal wall was sutured in three layers with the exception of a distance of 2 cm. through which a drain was passed to the line of intestinal sutures. A compressive dressing was applied.

On May 4, the patient was considered cured. He had a good appetite and digestion was easy. The stools were regular. The general condition was excellent and he had gained weight. Examination of the tissue showed that the cecum was congested; the wall slightly thickened, but there were no traces of ulceration of the mucosa. The ileocecal valve was soft. The three perforations were spread out over an extent of 6 cm., beginning at the origin of the colon. They were rounded and had a punched out appearance. The edges were slightly thickened and indurated. There were no traces of cancer or tuberculosis. The appendix was 8 cm. long, straight, regular in size and normal; its walls were soft. There were no adhesions, ulcerations or scars. The excised skin was thickened by a simple chronic inflammation. There was infiltration of leukocytes with bands of sclerotic connective tissue.

Case 30 (Zickler,[50] 1910).—*Perforation of the Ascending Colon.*

The patient was a woman, aged 25, without any previous history. She had had her first child a month previously. The confinement was normal. On May 25, while in the toilet, she had a sudden violent pain in the lower part of the abdomen which soon decreased, but later reappeared with greater intensity and was accompanied by vomiting. She entered the hospital thirty-six hours after the onset of symptoms. On admission, she had symptoms of peritonitis and diagnosis was made of appendicitis.

Laparotomy was performed on May 27. On account of the serious condition of the patient, nothing could be done except to remove the feces in the abdominal cavity and drain. Her condition improved a week after the operation. A second operation was performed on June 2. The pus pocket was cleaned, and a perforation

50. Zickler: Perforation of the Ascending Colon, Beitr. z. klin. Chir. **67**:189, 1910.

the size of a 5 franc piece was found. This perforation was almost round and was situated on the antero-external wall of the ascending colon and was parallel to the axis of the intestine. The mucosa was invaginated. The appendix was adherent, but did not show any perforation. The perforation was excised and sutured. The patient's general condition improved, but the local condition did not show any improvement. The third operation, a median laparatomy with a transverse incision, was performed on July 18. The ascending colon was freed of numerous adhesions. Resection of 10 cm. of intestine with terminoterminal anastomosis. She was cured on August 7.

CASE 31 (Lyle,[1] 1912).—*Simple Ulcer of the Colon; Perforation.*

A woman, aged 26, was admitted to St. Luke's Hospital in October, 1909, with the diagnosis of acute appendicitis, with abscess. The history and clinical symptoms on admission appeared to be those of an acute suppurative appendicitis engrafted on a chronic appendicitis. On admission, her temperature was subnormal and the pulse rate, 92. A blood count showed 25,000 white cells, with 86 per cent of polymorphonuclears.

An incision was made over the mass, and a large abscess containing fecal matter was evacuated. On the inner wall of the cavity, about 2 inches (5 cm.) above the cecum, there was a large, oval, indurated ulcer, in the center of which was a perforation, with necrotic edges. The edges were trimmed, a section removed for examination, and the perforation was closed. The appendix was outside of the cavity and apparently had nothing to do with the condition, but as it might have been the source of the infection, it was removed. A small rubber dam drain was inserted and the wound was closed. The patient made an uneventful convalescence, and left the hospital twenty-three days later. Microscopically, the appendix showed nothing abnormal, and the section from the ulcer showed it to be a simple ulcer of the colon.

On March 12, 1912, the patient returned to the hospital for the correction of a ventral hernia springing from the scar of the former operation. When this operation was performed by Dr. Gibson, the only evidences found of the former ulceration were a few fine adhesions on the colon. The picture presented by the original condition was so striking that one might have believed he was dealing with a gastric perforation. After the operation, a more detailed history was obtained, and it was then learned that the patient had long been a sufferer from indefinite gastric symptoms. She had made the rounds of the clinics, and her condition had been variously diagnosed as ulcer of the stomach, gallstones, floating kidney, chronic appendicitis and cancer.

CASE 32 (Eunike,[51] 1918).—*Simple Ulcer of the Colon.*

Louis Sch., aged 53, was admitted to the hospital on Feb. 28, 1918. His past history showed that an operation for double inguinal hernia had been performed several years previously, the right side being operated on first. The condition had recurred. The patient had always had difficulty in having bowel movements. He was often very constipated. Of late, he complained of severe pain in his abdomen. Eleven days before admission to the hospital, he began to have severe attacks of pain. His bowel movements were irregular and thin. For the past three days, he had been vomiting.

Physical examination revealed a strong, well nourished man, with a full, strong and regular pulse beat. Marked tenderness was elicited in an area over the

51. Eunike, K. W.: Ulcer Simplex of the Colon, Deutsche Ztschr. f. Chir. **147**:284, 1918.

abdomen, which was tympanitic. Both herniae recurred. On the right side, there was a small recurrence and a light, pale small operative scar. The left hernia was about the size of a man's fist and was irreducible and tense. A diagnosis of strangulated left hernia was made.

An operation was performed on the left hernia under local anesthesia. The whole sigmoid flexure was tied down with adhesions and the wall was markedly hypertrophied. The incision was enlarged, and gas anesthesia administered. On enlarging the incision, there was a fibrous exudate, the form and color of which resembled that coming from a perforated ulcer of the stomach. Under deep general anesthesia, the hernia was repaired and an incision was made from the xyphoid to the navel. Again appeared the fibrous exudate. The stomach was found to be normal, but on the hepatic flexure a perforation could be seen. The wall of the bowel was much infiltrated. The perforation was sewed with considerable difficulty. The ligatures were cutting through. Tamponade and drainage was instituted. The patient made a good immediate convalescence, but died on the fifth day after the operation. Autopsy showed general peritonitis. The sutures on the hepatic flexure held. In the close proximity of the perforation, there were three or four small ulcers. The rest of the colon did not show any signs of a pathologic process. The whole large intestine was hypertrophied.

CASE 33 (Soupault,[5] 1920).—*Perforation of a Simple Ulcer of the Colon.*

A woman, aged 22, was admitted to the hospital suffering from abdominal disturbances of acute onset. The diagnosis of appendicitis had been made, with the surmise that in the course of an acute attack with remission, perforation had occurred some hours before the patient's entrance.

Operation was performed, the Roux incision of 10 cm. being used. Incision of the peritoneum promptly revealed on the anterior aspect of the presenting coil, a perforation the size of a small pea, from which fecal matter escaped. The perforation was immediately closed with forceps, and the intestinal coil (cecum and ascending colon) was gently wiped off with a moist compress. The perforation was found to be situated on the antero-internal border of the ascending colon, 10 cm. above the cecum. The rounded perforation measured from 4 to 5 mm. in diameter. The borders were slightly thickened and ecchymotic. The surroundings were slightly swollen and edematous, but not indurated. The intestinal wall was flaccid and hyperemic. The peritoneum had lost its normal sheen and presented a few false membranes. The appendix, lying in the focus of peritonitis, was not seriously affected, but was resected. The perforated ulcer was rapidly sutured with linen thread purse-string suture; a second seroserous layer was closed by a U suture. The coils were irrigated with ether and two drains were placed in Douglas' space and in the iliac fossa, respectively. The wall was closed in two layers. The operative procedure took thirty minutes. The patient made a good recovery.

CASE 34 (Bazy,[52] 1920).—*Perforation at the Origin of the Ascending Colon.*

A man, aged 65, was brought to the hospital with symptoms of progressive intestinal occlusion. He had been having violent pains in the abdomen for eight days. He experienced severe colic which was generalized over the entire abdomen. He vomited. For two or three days, there had been the complete arrest of feces and gas. Interrogation of the patient showed that he had had intestinal disorders for a long time, and on several occasions he had presented painful crises accompanied by vomiting. He had chronic constipation. For these reasons, and considering the age of the patient, the physician who sent him to the hospital

52. Bazy: Bull. et mém. Soc. de chir. de Paris **46**:416, 1920.

believed that he had an intestinal obstruction in all probability due to cancer of the larger intestine. On admission to the hospital, he was in bad general condition; his complexion was earthy, his features drawn, and slight jaundice of the conjunctiva was noted. The pulse rate was about 100; the temperature, 38.5 C. (101.3 F.). Since his admission, the patient vomited several basins full of greenish fluid. His abdomen was distended, tense and was extremely painful throughout. The slightest pressure produced contraction of the muscles. There were no peristaltic contractions of the intestine. The arrest of feces and gas had been progressive and seemed to be total. It was thought that he had an occlusion due to cancer, but considering the condition of the patient, his temperature, the sharp pains on palpation and finally the appearance of the vomitus, it was suspected that this neoplasm was accompanied by inflammatory phenomena.

A median laparatomy was performed. When the abdomen was opened, it was seen that the intestinal loops were dilated, red and adherent to each other by means of fibrinous adhesions. The entire abdomen was carefully explored, and it was found that the right edge of the omentum was thickened and indurated and extended toward the right iliac fossa. It was adherent to the anterior wall of the ascending colon at its origin. It was carefully raised and was easily detached allowing the upper surface of the large intestine to be seen. Just above a line passing through the upper border of the intestine at its opening in the cecum, there was a regular circular perforation the size of a 50 centime piece. Its edges were sharp, hard and was located in the middle of a small indurated zone. This perforation was immediately closed with a tampon, and an incision was immediately made in the right iliac fossa, through which the portion of the large intestine on which the ulcer was situated was exteriorized. The exteriorization was maintained by fixing the intestine to the abdominal wall by means of several catgut sutures. This was done to gain time as the patient was already weak, and it was believed that the cecoiliac fistula thus established would have a good effect on both the peritonitis and on the intestinal obstruction. The intestinal loops were cleansed with ether through the median incision, and the operative incision was closed. A large drain was placed in Douglas' culdesac. Unfortunately, the patient died during the night following operation. The large intestine was removed at the autopsy. It presented no other lesion except the perforation which was situated at the origin of the colon on its anterior surface. The mucosa of the ascending colon and cecum seemed to be normal. With the exception of the induration which surrounded the ulcer, which was of slight extent, no other alteration of the intestinal wall could be found. The specimen was sent to a laboratory for examination, but unfortunately was lost so that no histologic examination could be made. Macroscopic examination showed that this perforation was distinctly different from those seen in ordinary ulcerations of the intestine and no lesion was found which could explain its production.

CASE 35 (Idem).—*Perforation of the Cecum.*

A youth, aged 20, was admitted to the hospital on Jan. 20, 1903, for appendicitis. He was operated on for an abscess of appendicular origin on February 25. A right lateral incision was made along the external border of the right rectus muscle. As soon as the peritoneum was seen, it was evident that pus would be found; in fact, on opening the abdomen, a creamy greenish pus with fecal odor escaped. After having sponged this pus, it was found that the intestinal loops were adherent to each other. The hand was passed along the abdominal wall until the posterior wall of the cecum was reached. At this time bubbles of gas

were seen to arise, and at the same time the characteristic noise of intestinal perforations was heard. The appendix was twisted on itself and adherent, but there were no special lesions. On the other hand, a certain distance away from it on the anterior surface of the cecum was found a circular perforation which easily admitted the tip of the index finger. This perforation was easily closed by a double line of silk sutures. The appendix was removed. A drain was left in the abdominal cavity. The abdominal wall was closed in three layers. The patient left the hospital, cured, on April 7, 1903.

Case 36 (Gregoire,[53] 1920).—*Perforation of an Ulcer of the Sigmoid.*

A man, aged 55, of robust constitution but with chronic constipation, had passed stools on several occasions which contained blood. He attributed this to hemorrhoids. On June 1, 1919, while walking, he had pains in the lower part of the abdomen and was obliged to go home. He thought that constipation was the cause of the pain, and he took an enema. When he went to the toilet, he suddenly had a violent pain with vomiting. There was violent colic. He went to the hospital seven hours later. On admission, he was in great pain but his general condition did not seem to be bad; the temperature was 37.8 C. (100 F.); the pulse rate, 104. His abdomen was greatly distended and painful throughout. There was an absolute arrest of feces and even of gas. On account of the sudden onset of these peritoneal symptoms, it was thought that he had acute appendicitis with general peritonitis. Operation was performed seven and a half hours after the beginning of the symptoms.

A right iliac laparotomy was performed. On opening the abdomen, a rather large quantity of grayish fluid mixed with fragments of feces was discharged. However, nothing was found at the ileocecal angle or at the level of the appendix. A small amount of fecal material was found in the true pelvis. The iliac incision was then lengthened downward and inward, and the epigastric artery was ligated. The true pelvis could then be easily explored. It was literally filled with an enormous quantity of hard and pasty feces. After cleaning out the pelvis, a perforation the size of a 50 centime piece was found on the anterior surface of the sigmoid. The edges were slightly indurated for a short distance, and beyond the induration the mucosa of the colon was apparently normal. The edges of the perforation were resected, and the loss of substance was sutured in two layers. Although the peritoneum and the intestine did not appear to be inflamed, extensive drainage was inserted on account of the presence of such a large amount of feces in the abdomen.

During the following days, the patient seemed to support the operation well; there was no vomiting, his general condition was good, and he passed gas spontaneously. However, there was abundant suppuration from the abdomen. It was later learned that on June 16, this man again had abdominal pains with profuse vomiting which became fecaloid, and he died. It was believed that these symptoms were due to probable occlusion. Autopsy was not performed.

Case 37 (Sieur,[54] 1920).—*Perforation of the Cecum.*

The patient was a man about 30 years of age. When the author saw him he had an acute abdominal attack which was so serious that it was thought advisable to send him to the hospital at once. The surgeon at the hospital did not believe that he would be able to operate and the patient died three days later. At autopsy, a perforation of the cecum was found. It was the size of a 50 centime piece and

53. Gregoire: Bull. et mém. Soc. de chir. de Paris **46**:420, 1920.

54. Sieur: Bull. et mém. Soc. de chir. de Paris **46**:421, 1920.

was situated 3 cm. above the insertion of the appendix. The appendix was normal as well as the rest of the large intestine. The perforation had allowed fecal matter to pass into the peritoneum, and especially beans of which the patient had eaten a large quantity twenty-four hours before the beginning of symptoms. The edges of the ulcer were indurated and it had the appearance of an old lesion. The patient was a heavy eater and had chronic constipation. For three months he had been having discomfort in the right iliac fossa.

CASE 38 (Lardennois,[55] 1920).—*Spontaneous Perforation of the Cecum.*

A man, aged 52, gave a past history which showed that he had had lead colic during the previous year, which had appeared while he was working on storage batteries. In addition, he presented evident signs of lead poisoning. Later, he suffered from digestive disorders and anorexia, difficult digestion and constipation. On September 9, while walking to work, he suddenly had a severe pain in the right iliac fossa and had to be taken home. A short time later, nausea and vomiting occurred. According to the patient's story, the right iliac fossa was extremely painful and the vomiting persisted. There was no evacuation of feces or gas. His condition improved slightly after several days, and on September 12 he took a purgative. He had several stools following this purgative, but the symptoms rapidly became worse and he was brought to the hospital during the night of September 16 in a dying condition. On admission, it was found that he had peritonitis. A median laparotomy was performed. Cloudy fluid was found in the entire abdomen. There were abundant false membranes. There was a circular perforation with sharp edges about 5 mm. in diameter on the cecum. It was at the most protruding portion of the antero-internal wall and appeared to be larger at the level of the mucosa than at the serosa. The appendix seemed to be normal. The cecal opening was rapidly closed. The appendix was removed for examination. Inspection of the abdomen did not show any other lesion. There was no other ulcer, nor was cancer perceptible on careful examination. The adhesions were liberated, and the intestines were washed with ether. Drainage was instituted. The patient died during the course of the following night.

A later examination showed nothing more than was found during the operation. The appendix did not present any lesion other than those of superficial congestion due to its presence in an area of peritonitis. Histologic examination was not made.

CASE 39 (Schoemaker[56]).—In 1906, the author treated a man who had been sick for five days. The patient had always been in good health previous to this time. On Sept. 12, 1906, he suddenly had a sharp pain in the abdomen which was followed by vomiting. At first the pain extended over the entire abdomen, but little by little it became localized in the lower part. The pain diminished in intensity while his general condition became worse. He was sent to the hospital on September 17. On admission, his appearance was bad. He had a characteristic peritoneal facies. Rectal examination showed that Douglas' pouch was distended and painful. The diagnosis was acute appendicitis (low appendix which had perforated and had given rise to an abscess in the true pelvis).

An incision was made in the median line from the symphysis almost to the umbilicus. On opening the peritoneum, a large amount of fetid pus escaped. The cavity was emptied and dried with gauze tampons. The loops of small intestine were adherent to each other. The appendix was found. It was red like the intes-

55. Lardennois: Bull. et mém. Soc. de chir. de Paris **46**:447, 1920.

56. Schoemaker, quoted by Soupault.

tine, but it was no more inflamed than the intestine and there was no perforation. A small round hole was found in the sigmoid from about 3 to 5 mm. in diameter, and about 12 cm. above Douglas' culdesac. The edges were infiltrated, and its appearance was that of a perforated ulcer of the stomach. The edges were excised, and a small wound in the intestine was closed. Drainage was established and the abdomen was partially closed.

The patient went into collapse on September 18. His pulse was scarcely palpable, and in spite of stimulants, he died in the evening.

Autopsy revealed normal thoracic organs. The stomach and the small and large intestines were opened. No inflammation of the mucosa or ulcer was found. The mucosa near the small intestinal wound in the colon was normal. Microscopic examination of the edge of the ulcer showed an infiltration of white cells. There was no evidence of tuberculosis.

CASE 40 (Levy,[57] 1921).—*Simple Ulcer of the Colon.*

E. F., a servant girl, entered the hospital on April 9, 1920. There was no history of tuberculosis in the family. She always had enjoyed good health until three years before, when she began having "stomach pain" lasting from one to two hours, which was so slight that she paid little attention to it. In September, 1919, she was treated for the first time in a hospital for pain that came on suddenly in the right side of the abdomen. She was discharged in ten days. She was admitted to the hospital again in January, 1920, for the same symptoms. Since that time she has frequently suffered from constipation and had to take purgatives. Three days before her present illness, she complained of dull pain over her whole abdomen which decreased a little after the passage of gas and a single stool, but the day before admission increased and became localized in the right side of the abdomen and from that time on was continuous. She did not suffer from vomiting. A hard stool was passed spontaneously in the night with severe pain. After that, the pain decreased considerably, but began again after about three hours.

Examination on admission, on April 9, 1920, revealed a large, strong girl in an excellent condition of nutrition. The left half of the abdomen was soft, without rigidity and without pain, and was compressible. On the right, a little inward from McBurney's point a rather long, oval, moderately hard tumor the size of a small hen's egg could be felt; it was movable and sensitive, particularly in the central part. There was no abdominal rigidity on the left and a trace on the right. Gynecologic examination revealed the uterus retroflexed, and no swelling of the adnexa.

The symptoms seemed to suggest appendicitis, though the symptoms on palpation left serious doubt of the diagnosis. The movable tumor inward from McBurney's point did not fit into the picture of appendicitis, in which the tumor, if there is any, is generally fixed firmly in the hollow of the sacrum. But operation seemed to be indicated.

Operation (Prof. Alsberg) was performed under general anesthesia. When the abdominal cavity was opened, turbid-serous fluid gushed out. A hard tumor could be felt on the wall opposite the ileocecal valve when the cecum was pulled out; it was about 3 by 6 cm. in size, long, hard, smooth and a little indented in the middle. Its red color was distinctly different from the velvet-red of peritonitis and showed dilatation and branching of the vessels. The indentation in the

57. Levy, Alfred: Simple Ulcer of the Colon, Deutsche Ztschr. f. Chir. **165:** 366, 1921.

middle of the tumor proved to be an area about the size of a 2 mark piece covered with a fibrin deposit. On bimanual palpation, from the lumen of the intestine through an invagination in the wall of the cecum, this area was found to be a crater-shaped, rather long oval, sharp-edged ulcer, the inner and outer finger tips being separated from each other by a thin layer. The appendix did not show any pathologic change.

An appendectomy was performed. The pathologic part of the wall of the cecum was invaginated and buried in two stages by serosa sutures so that normal serous surfaces came together. Suture of the wall of the abdomen was done in two stages, without drainage. On April 16, the wound healed by first intention. Bowel movements were obtained only after enema. The patient's condition was good. In the histologic specimen of the appendix (serial sections), no pathologic changes were noted, particularly no tuberculosis. On April 25, the patient was up. Enemas were still necessary for bowel movements. Repeated examination of the stools for tuberculosis bacillus were negative. On May 11, a stool was passed after a purgative without any symptoms. The patient weighed 129 pounds (58.5 Kg.). She was discharged from the hospital cured.

CASE 41 (Ecot and Richard,[35] 1922).—*Perforation of a Simple Ulcer of the Ascending Colon.*

This recent observation on a perforation of a simple ulcer of the ascending colon, seen by the authors in the Service of Professor Pieree Duval, is of particular interest from the clinical point of view (syndrome of occlusion) as well as the anatomic point of view, the only one here considered. The specimen was removed in the course of the operation and showed a rounded, punched-out perforation of from 6 to 8 mm., with thickened margins, situated on the antero-external aspect of the ascending colon, 10 cm. above the ileal opening. The perforation permitted the escape of fluid material and gas; the patient at the time was suffering from perforation peritonitis. Excision of the ulcer, suture of the colonic wound and drainage were followed by recovery. Direct polymorpho-nuclear infiltration around the glands was visible at one extremity of the specimen. Embryonic infiltration was noted, especially abundant at the level of the muscular mucosa. Diffuse sclerosis, separating the fibers of the muscular mucosa layer, and perivascular sclerosis were also present, but no endarteritis. The capillaries were dilated and there was diffuse hemorrhagic infiltration. The serosa was slightly thickened. Accordingly, the condition was evidently long-standing mild sclerosis of the colon, apparently simple, as specific lesions of syphilis, tuberculosis or cancer, were not demonstrable at any point. The course of the disease became complicated in the following days by intestinal obstruction. An artificial left iliac anus was made.

CASE 42 (Dickinson,[3] 1922).—*Perforating Ulcers of the Cecum.*

A man, operated on, April 3, 1895, had had acute right inguinal pain and tenderness. There was no tumor. The rectal temperature was 101 F. The condition was diagnosed as appendicitis. A right rectus incision was made. The appendix was normal. The cecum was enormously distended, with three points of impending perforation 6 cm. in diameter. The peritoneum was reduplicated over these. Necropsy, five days later, revealed that the points of suturing held, but there were six new perforations in the cecum. These ulcerations were punctate, round, sharp margins, not necrotic.

CASE 43 (Idem).—A man, aged 22, operated on, Dec. 4, 1916, had suffered for a number of years with gassy dyspepsia, sour brash and delayed digestion, with relief on vomiting and never any pain. There was some discomfort in the

appendicular region. The condition was diagnosed as chronic appendicitis or cholecystitis. The gallbladder was opened, disclosing dark bile, thickened with mucus, but no calculi. Drainage was instituted. The remainder of the abdomen was apparently normal on palpation and exploration, with the exception of the appendicular region. A second opening was made. The appendix was clubbed, fibrous, and excised through the tip of the cecum. On manipulation of the cecum to explore adhesions, a ligature in the cecal incision pulled off, with discharge of a substance resembling a dysenteric stool. The stoma was religated with linen thread. Necropsy, on the fourth day, revealed the ligature on the appendix stump in place. There was a ragged perforation of the cecum the size of a 5 cent piece opposite the ileocecal junction.

Case 44 (Idem).—A man, aged 56, operated on, July 21, 1921, a clerk of good habits, experienced abdominal trouble three weeks before, starting with severe pain at the umbilicus, and green, watery vomitus. This recurred with intermissions. During the second week of the present illness, the pain shifted to the right lower quadrant. Vomiting continued, the vomitus becoming brown. The patient had been given considerable bismuth. Roentgenograms were taken. There had been no action of the bowels in ten days. On admission to the hospital, the patient presented marked pyorrhea, coated tongue and reddened throat. Pain, rigidity and tenderness were noted at the cecal region, particularly on deep pressure. The heart sounds were distinctly heard at the pubes. As some type of appendicitis was suspected, an incision was made through the right rectus sheath. When the peritoneal cavity was opened, a puff of gas was heard. The peritoneum and viscera were markedly injected, and purplish red. The appendix was normal. The cecum was agglutinated to the lateral wall. When it was lifted up, liquid feces exuded from several small openings in the ulcerated areas, about opposite the ileocecal junction. These points of ulceration were about 6 cm. in diameter. In view of previous experiences, the cecum was stitched to the abdominal peritoneum and incised freely. Bismuth feces were removed. Convalescence was protracted, and during it the patient developed exfoliated stomatitis, associated with which was complete anorexia and an intolerance by the stomach even for water, except in small quantities.

Case 45 (Bowen,[58] 1922).—*Perforation of the Large Bowel.*

A woman of middle age with a negative gastric history was taken suddenly with severe abdominal pain, nausea and vomiting. Eight hours later, the abdominal wall was rigid and the signs indicated a perforation. Operation revealed fecal material in the abdomen, and a perforation of the sigmoid. The walls of the colon were described as rotten, and they only tore on attempts to suture. The woman died, but autopsy was not permitted. The author was unable to get a clear idea of the abdominal condition, but thinks the perforation probably resulted from thrombosis. Evidence in favor of a stercoral ulcer or foreign body was scanty.

Case 46 (Idem).—A woman, aged 27, who had been suffering from gastric ulcer, developed severe pain all over the abdomen, with a definite right sided rigidity, and maximum tenderness just above and internal to the anterior superior spine. Operation revealed an acute catarrhal appendicitis, with a perforation in the cecum, near the ileocecal valve. There was no free fluid in the abdomen, the perforation having been closed by some valve action. As no constipation had been present, a stercoral ulcer can be ruled out.

58. Bowen, W. H.: Five Cases of Perforation of the Large Bowel, Guy's Hosp. Rep. **72**:441 (Oct.) 1922.

CASE 47 (Chalier and Mallet-Guy,[59] 1923).—*Simple Ulcer of the Ascending Colon.*

A man, aged 50, was admitted to the hospital on Oct. 3, 1921, with evidence of active pulmonary tuberculosis. Nothing else abnormal was found, and there was nothing to indicate any disorder of the digestive tract. This condition persisted without any marked change up to Jan. 23, 1922, with a temperature ranging between 99.6 and 101.1 F. On this date, diarrhea occurred without any previous colic and there was an intestinal hemorrhage. There were four stools of red blood without clots amounting to about 1 liter. At the same time his temperature went down to 98.9 F. and the patient became pale and enemic. He complained of vertigo and marked asthenia. The hemorrhage was relieved by rest, diet, applications of ice to the abdomen, by the administration of small, very hot enemas and the administration of calcium chloride and epinephrine. Several days later, when all danger seemed to be over, a careful and complete examination of the patient was made to determine the cause and location of the hemorrhage.

The respiratory symptoms were not changed. Rectal examination was negative. No abdominal tumor could be palpated, but the spleen was increased in size and extended about 2 inches beyond the costal border. In addition, there was dulness in the flanks which moved with a change in position and showed the presence of a slight degree of ascites. The intestinal hemorrhage was not repeated, and one and a half months before death he began to complain of pains in the umbilical region. He had a permanent burning sensation which was better after taking food. Three hours later there would be a marked increase of the pain. At this time the pains would be sharp, radiating to the left shoulder and persisting for two or three hours. They never terminated in vomiting, but were relieved by evacuation of the bowels. Diarrhea generally occured. There were five or six stools daily, sometimes preceded by slight colic. His appetite was fair. Palpation was painful in the right iliac fossa. It was even more painful in the epigastric region just below the xyphoid and no tumor could be felt. His general condition became worse and it was not believed advisable to cause him to undergo the fatigue of a x-ray examination. Being confined to bed with his diet restricted, he rapidly became cachetic and he died on April 8, 1922.

At autopsy, the lungs showed evidence of tuberculosis. The right suprarenal capsule, to a large extent, showed caseation. The left suprarenal was normal. On the ascending colon about 5 cm. above the ileocecal valve, there was found on the antero-external surface an ulceration arranged transversely, perpendicular to the axis of the colon. This ulceration was of linear form about 1 cm. long and from 1 to 2 mm. wide. The edges of the ulcer were slightly elevated and were dark. The base of the ulcer appeared to reach the subserosa. There was no peritoneal reaction at this level. On examining the serosa, a small vessel could be seen by transparence. It was in a direction perpendicular to the axis, and a part of its course was exactly in relation to the base of the ulcer. This vessel was obliterated at a point in its course which corresponded to the center of the ulcer; it was undoubtedly this vessel which gave rise to the intestinal hemorrhage. The entire mucosa of the ascending colon was pink, while this coloration was absent in the rest of the intestine. The mucosa of the large intestine presented many folds without any appreciable lesions. No ulcer or other lesion was found on the small intestine, duodenum or stomach.

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59. Chalier, J., and Mallet-Guy: Simple Ulcer of the Ascending Colon, Arch. d. mal. de l'appar. digest. **13**:521 (June) 1923.

Histologic examination revealed a simple ulcer of the colon without any tuberculous or neoplastic formation. The edges of the ulcer were raised. The ulcer had extended through the muscularis mucosa and had deeply penetrated the muscular layer. This progression was accompanied by a marked lymphocytic reaction. The vessels of the subserosa were obliterated. The right suprarenal was caseated, but there were some traces of glandular tissue. Giant cells were found. The section included a sympathetic ganglion, which was normal.

Case 48 (Mintz,[28] 1923).—*Simple Ulcer of the Colon.*

A girl, aged 18, took sick with vomiting and abdominal pains, evacuation of fifteen nematodes took place five days after castor oil had been given. Within the next eleven days, intermittent attacks of colic and constipation occurred. On the sixteenth day, the attending physician established evidence of a mobile tumor in the ileocecal region and prescribed oleum chenopodii, whereupon another ascaris was evacuated. On the seventeenth day, during examination in my department, an extremely mobile tumor was found below the umbilicus. The diagnosis of chronic invagination was confirmed by the x-ray examination. Seven hours after ingestion of the contrast meal, the lower ileum and also part of the ascending colon were filled, and the invagination of the ileum loop could be directly seen on the plate. Stools that resembled the fecal matter of sheep were passed on the same day. On the evening prior to operation (twentieth day of the disease), the colicky pains, which had been constant and slight, became more severe. The tumor became enlarged to a cylinder which disappears obliquely upward to the left underneath the costal arch. Later on, vomiting set in. Immediate surgical intervention was advised. The ileum, cecum and ascending colon were invaginated into the transverse colon. Disinvagination of the edematous intestinal parts was performed without difficulty. In the cecum, a hard indentation of the wall, opposite the orifice of the ileum was noticeable. This was the size of a 1 mark piece. The serosa over it was thickened and flaming red—in short, the picture was presented that one sees on the serosa over peptic ulcers. Typical ileocecal resection was performed. The patient had an uneventful postoperative course.

The cecal wall as a whole was unchanged. The indentation appeared to have been due to the inflexibility of the diseased place in contrast to the distended, healthy adjacent parts. On the mucous membrane, there was a sharply defined ulcerated surface of the size of a 1 mark piece which corresponded to the indentation. This surface was of brownish red and contrasted markedly from the completely healthy appearing adjacent mucous membrane. A similar ulcer the size of a pea was situated 1 cm. away from it, at the same level.

Microscopic examination revealed the serosa considerably thickened, with newly formed dilated vessels. The larger ulcer showed a sharp demarcation from the surrounding area, loss of mucous membrane, small celled infiltration and scattered necrotic foci, which are also found in the muscularis. On the little ulcer, similar conditions were found which did not penetrate so deeply. Thrombosing endarteritis could not be demonstrated.

Case 49 (Broere,[60] 1923).—*Decubitus Ulcer of the Colon.*

This contribution comes from the Pathologico-Anatomical Laboratory of the Wilhelmina Institute in Amsterdam, and concerns the autopsy observations in the case of an old woman 80 years of age. An ulcer was found at the transition of the rectum into the sigmoid flexure of the colon. Rupture of a so-called decubitus

60. Broere, W. J.: Decubitus Ulcer of the Colon, Nederl. Tijdschr. v. Geneesk. **67**:2274, 1923.

ulcer was caused by a fecolith. The perforation orifice was found to contain a hard fecal mass. The symptoms observed during life, at the time of perforation, in the form of profuse hemorrhage from the anus and sudden onset of pain, were explained by the (microscopically) distended and congested vessels, which extended almost to the intestinal lumen. In spite of her advanced age, the patient had been in good condition up to a few months before her admission to the hospital on account of general weakness. As she suffered from chronic constipation, cascara was regularly administered. Her condition some months later was entirely satisfactory, when she was suddenly attacked by severe abdominal pains. The abdomen was painful on palpation, with some muscular defense. On three occasions, the patient passed a few small scybala with about 75 and 100 cc., respectively, of fluid, unchanged blood, without mucus. The results of the rectal examination were negative. At night, her hands became cold and the heart sounds became weaker; the pulse rate was 120 and weak. In the course of the next day, the temperature rose, the condition became aggravated, the pulse rate increased to 160, was weak and small, and at the end of the second day after the onset of the symptoms of perforation, the patient died. The autopsy explained all symptoms, indicating that the microscopically greatly congested vessels, reaching nearly to the intestinal lumen, were responsible for the profuse hemorrhages from the anus at the time of the perforation, with simultaneous onset of severe pains.

CASE 50 (Boss,[21] 1927).—*Perforating Ulcer of the Large Intestine.*

A man, aged 38, entered hospital on Aug. 8, 1921. Eleven years before he had had gastro-intestinal catarrh while in Arabia which lasted eleven days; ten years before he had had malaria in India. There were no further intestinal disorders. On August 7, the afternoon previous to his admission, while in perfect health, he had had sudden, violent, convulsive pain in the umbilical region and vomiting. On August 8, the day of admission, he passed a black stool. Vomiting and pain persisted in spite of the administration of narcotics. Peritonitis was suspected, and he was sent to the surgical ward of the hospital. On admission, physical examination showed him to be a robust man, in fair condition of nutrition, healthy appearance of skin and face. The blood picture revealed 11,200 leukocytes with 90 per cent polymorphonuclears. The temperature was 102 F.; pulse rate, 110, regular and full. The results of the physical examination were negative except that in the abdomen, the scaphoid was indrawn; there was intensive resistance, and the greatest pressure pain in the region between the navel and the symphysis. The urine was normal. A diagnosis of peritonitis was made, and operation was immediately performed.

A median laparotomy was performed. The small intestines were reddened. There was some pus deep in the pelvis. It was more abundant in the median line than in the region of the appendix. The appendix was free and was found not to be responsible for the peritonitis. The intestines in the pelvis were covered with fibrin. In the lower portion of the sigmoid, there was a perforated ulcer the size of a bean from which feces were discharged. The tissue in the vicinity of the ulcer was not infiltrated but reddened. The ulcer was closed by five longitudinal catgut sutures and two rows of catgut sutures. To relieve the strain on the sutures, a broad colostomy was made in the descending colon, three handbreadths above the suture line.

Two days after operation, there was a large stool from the fecal fistula. The patient made an uneventful convalescence and was discharged as well on December 15. Four weeks after operation sigmoidoscopy revealed a normal condition of the mucosa for 22 cm. The ulcer had disappeared.

Roentgen-ray examination of the intestine after a barium meal and a barium enema showed that in passage from the descending portion of the large intestine into the sigmoid flexure, a constriction occurred, lasting several hours, at a point which corresponded to the location of the sutured ulcer. The following history was obtained from the patient some time after his operation: Six months before the perforation, the patient had had an accident while carrying a heavy load. He had slipped and fallen, injuring his back. From that time on he had had considerable abdominal pain when lifting heavy weights.

Cases 51, 52 and 53 are my cases 1, 2 and 3 which have been reported earlier in this paper.

"STREAM LINE" PHENOMENA IN THE PORTAL VEIN AND THE SELECTIVE DISTRIBUTION OF PORTAL BLOOD IN THE LIVER *

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There have been a few experimental and clinical studies that have suggested the possible occurrence of a selective distribution of portal blood in the liver and the presence of independent currents of blood in the portal vein. Final proof of the presence of these currents has not been established, and the selective distribution of portal blood in the liver has been held in doubt. The possible existence of segregated streams of blood in the portal vein and their subsequent distribution to definite parts of the liver is of interest and should be of considerable practical significance in the study of the physiology and pathology of the liver. The evidence in favor of the existence of these phenomena has not generally been known or seemingly accepted.

In 1901, Sérégé [1] injected india ink into the splenic vein of the dog and found particles only in the left lobe of the liver; when the ink was injected into a tributary of the large mesenteric vein, particles were found only in the right lobe. These experimental observations were confirmed by Glenard.[2] Bauer [3] and several other workers repeated the experiments, but they were unable to distinguish any difference in the distribution of the india ink, and therefore denied the existence of a dual portal current. In 1907, Loeb [4] found that the iodine content of the right lobe of the liver of rabbits was constantly greater than that of the left one hour after subcutaneous injection of potassium iodide. This observation was confirmed by Wells [5] and Hedenburg.

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1. Sérégé, H.: Contribution à létude de la circulation du sang porte dans le foie et des localizations lobaires hepatiques, J. de méd. de Bordeaux **31**:271, 291 and 312, 1901.

2. Glenard, F.: Note sur les localizations lobaires hepatiques, Bull. et mém. Soc. méd. de hôp. de Paris **18**:386, 1901.

3. Bauer, A., and Brissant, E.: L'independence des lobes du foie est hypothèse, J. de l'anat. et de la physiol. **45**:1, 1909.

4. Loeb, O.: Die Jodverbeilung nach Einfur verschiedener Jodverbindungen, Arch. f. exper. Path. u. Pharmakol. **56**:321, 1907.

5. Wells, H. G., and Hedenburg, O. F.: Studies in the Biochemistry and Chemiotherapy of Tuberculosis: The Permeability of Tubercles for Iodin Compounds and Proteins, J. Infect. Dis. **11**:349, 1912.

In 1914, Bartlett,[6] Corper and Long studied the problem. Olive oil emulsified in blood serum was injected into different parts of the portal system of the dog. After counting the number of fat emboli in microscopic sections of different parts of the liver, they concluded that a dual portal current exists in the dog's portal vein; and that blood from the stomach, spleen, duodenum, first portion of the jejunum and rectum flowed mainly to the left lobe of the liver, while blood from the lower portion of the jejunum, the ileum and the first portion of the large intestine flowed mainly to the right lobe.

More recently, McIndoe[7] and Counseller made anatomic studies of the liver in man by means of the celloidin-corrosion method; these studies indicate an anatomic division of the liver into two separate units. These workers have demonstrated that the area of distribution of the right and left branches of the portal vein in the human liver is sharply divided along a line from the fossa for the gallbladder to the entrance of the hepatic veins into the inferior vena cava, and that the only communication between the two vessels is through intercellular sinusoids. The same line of demarcation separates the field of supply of the branches of the right and left hepatic artery and also the branches of the two hepatic ducts. The line of lobar separation, as determined by the celloidin method, corresponds to the recognized embryologic boundary between the right and left lobes of the liver.

In the present investigation, we made a study of the blood currents in the portal vein itself, and observed the subsequent distribution of the blood in the lobes of the liver. Dyes were injected into different radicles of the portal vein of the dog, and their ultimate localization in the liver was observed. The advantages of this method are that the injected material can immediately be visualized in the part of the liver to which it has been conveyed, and that the dye-stained blood can be seen in the portal vein when the vessel is submitted to powerful trans-illumination. The method overcomes the objections to the use of particulate matter for intravenous injections.

At the outset of this experimental study, emulsified iodized oils were introduced into various parts of the portal circulation, and roentgenograms of the liver were obtained. An unequal distribution of the iodized oil was often noted, but the site in the liver in which the oil localized itself was inconstant.

As we believed anatomic considerations would be of importance in determining the way in which a system of stream-lines might arise

6. Bartlett, Corper and Long: The Independence of the Lobes of the Liver, Am. J. Physiol. **35**:36, 1914.

7. McIndoe, A. H., and Counseller, V. S.: Bilaterality of the Liver, Arch. Surg. **15**:589 (Oct.) 1927.

and be maintained in the portal vein, we made a careful study of the portal system and its tributaries and also of the mode of termination of the portal vein in the liver.

THE ANATOMY OF THE PORTAL VEIN IN THE DOG

Mode of Termination of the Portal Vein at the Hilum of the Liver.—At the hilum of the liver, the portal vein divides into two main branches; the division takes place at an angle of about 170 degrees. The right branch of the portal vein is smaller and has a much shorter course than the left branch.

The right branch of the portal vein is distributed entirely to the two lobes of the liver which lie most laterally. After a short course outside the liver, the vessel divides into two or four branches, and one, or a pair of these, passes into each of the two right outer lobes.

The left branch of the portal vein traverses the hilum of the liver for a distance of about 5 cm.; in its course it gives off three branches which come off at right angles to it and pass directly to the three intermediate lobes of the liver; some of these branches may be paired. The main vessel ends by dividing in a radiate manner into five branches; each of the two large left lobes receives two branches, while the fifth branch passes into the small lobule of liver which is situated immediately to the left of the gallbladder; this lobule also receives a branch from the main left trunk (figs. 1 and 2).

It will be observed that strict *anatomic* bilaterality of the portal vein does not obtain in the dog's liver as it does in that of the human subject. In the dog, the left branch of the portal vein supplies two thirds of the whole organ; the right branch of the portal vein delivers blood only to the two outer right lobes of the liver.

Intrahepatic Course of the Portal Vein.—The intrahepatic course of the portal vein was studied in specimens in which iodized oil (40 per cent) had been injected into one of the tributaries of the portal vein in a living dog. Roentgenograms of the excised livers were taken, and an exact outlining of the venous channels was obtained (fig. 2).

It was observed that the main vein of each lobe splits up in an arborescent fashion, and that there is no anastomosis between the larger branches. The larger branches of the portal vein and their smaller ramifications are clearly displayed in figure 2. When livers from human beings had been injected with iodized oil 40 per cent a similar distribution of the portal vessels was demonstrated; furthermore, the strictly sectorlike distribution of the large portal branches suggest that the human liver may be regarded as a multilobar organ resembling closely the dog's seven lobed liver.

The Anatomy of the Portal Vein and of Its Tributaries.—In the dog, the average length of the portal vein is 8 cm. It begins dorsal to the neck of the pancreas by the union of the splenic and large mesenteric veins. The vessel runs in a cephalad direction, dorsal to the superior part of the duodenum, between the two layers of the lesser omentum to the right end of the porta of the liver, where it divides into a right and a left branch.

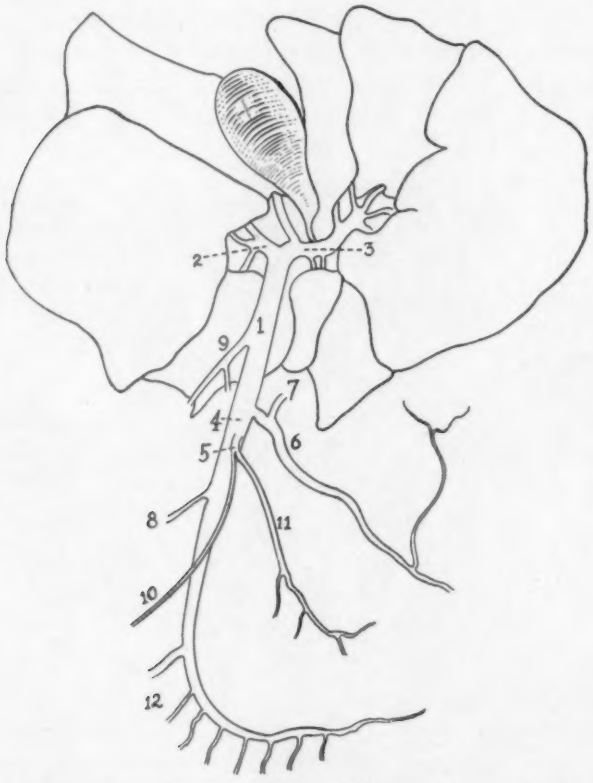

Fig. 1.—Diagram of the portal system of the dog showing the main tributaries and the mode of termination of the vessels at the hilum of the liver. *1* indicates the portal vein; *2* and *3*, the right and the left branch of the portal vein, respectively; *4*, the main mesenteric vein; *5*, the small mesenteric vein; *6*, the splenic vein; *7*, the gastric branch of the splenic vein; *8*, the lower pancreaticoduodenal vein; *9*, the upper pancreaticoduodenal vein; *10* and *11*, the right and the left branches of the small mesenteric vein and *12*, the enteric branches of the large mesenteric vein.

Tributaries: The chief tributaries are the splenic, the large mesenteric and the upper pancreaticoduodenal vein.

Splenic Vein: The splenic vein runs to the right from the hilum of the spleen and ends by joining the large mesenteric vein to form the

portal vein; it joins the mesenteric vein at an angle of about 45 degrees. About 2 cm. from its termination, it receives a branch from the posterior part of the stomach and lesser curvature.

Large Mesenteric Vein[8]: The large mesenteric vein is larger than the splenic vein; it drains blood from the lower part of the duodenum and the whole of the small intestine. Near its termination it has a linear course, and the portal vein appears to be a direct continuation of it.

The small mesenteric vein[8] enters the large mesenteric vein at an angle of about 5 degrees, less than 1 cm. below the point of union

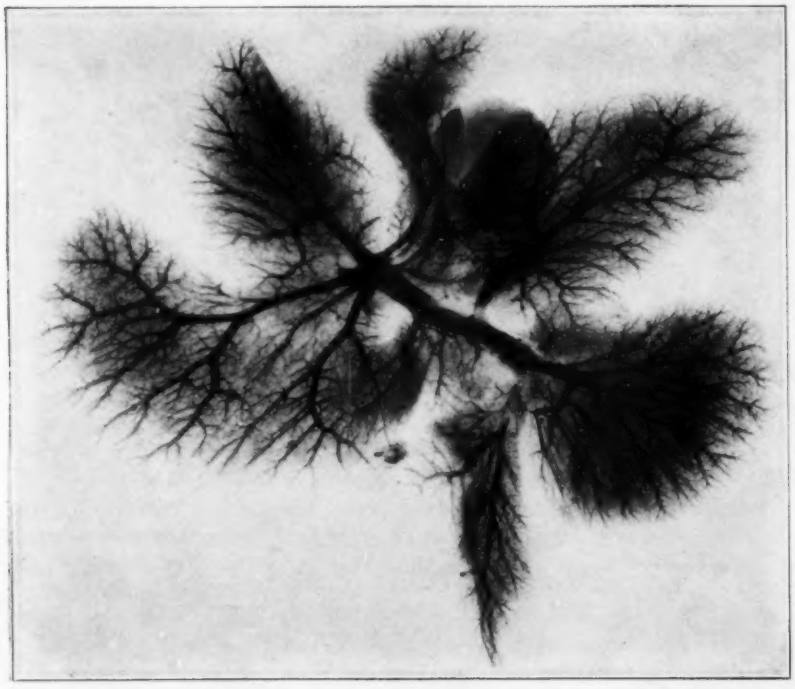

Fig. 2.—Dog's liver after intraportal injection of iodized oil. Note the arborescent character of the large portal veins and the absence of large intralobar anastomoses.

of the splenic and large mesenteric trunks. The small mesenteric vein drains blood from the whole of the colon; it has two main branches. The right branch comes from the cecum and the first part of the colon; the larger left branch collects blood from the distal part of the colon and rectum. The two branches unite into a short common stem that

8. In works on anatomy of the dog the mesenteric veins are called large and small. The large vein corresponds to the superior vein of the human subject; the small vein corresponds to the inferior mesenteric vein.

enters the left side of the large mesenteric vein a little toward its ventral aspect. In some dogs, the right and left branches of the small mesenteric vein do not unite to form a common vessel, but enter the large mesenteric vein separately at the same level. In such instances, the branch from the left portion of the colon enters on the left side of the large mesenteric vein, and the right terminates about the center of the ventral aspect of the main mesenteric trunk.

Pancreaticoduodenal Veins: There are two of these vessels. The upper pancreaticoduodenal vein drains the head of the pancreas and the first part of the duodenum and also receives a small tributary from the pyloric area. The vein ends by joining the portal vein on its right side, about 2 cm. below the hilum of the liver. The lower pancreaticoduodenal vein derives blood from part of the head of the pancreas and the distal part of the duodenum; it ends by joining the right side of the large mesenteric vein about 5 cm. from the termination of this vessel.

EXPERIMENTAL STUDY OF THE DISTRIBUTION OF THE PORTAL BLOOD CURRENT IN THE LIVER

Method.—Trypan blue, dissolved in blood serum, was employed as the coloring agent for the study of the portal blood current. One gram of trypan blue crystals was dissolved in 15 cc. of blood serum; 3 cc. of this solution was the amount generally given at each intravenous injection of the portal radicles; the quantity was varied slightly according to the size of the dogs employed. The advantages of the dye are its lightness and solubility, and the parts of the liver to which it is conveyed by the blood stream are immediately colored. The staining of the liver persists for some time, and formaldehyde preservation of the specimens does not obliterate the coloring.

The dogs were anesthetized with ether, the abdomen opened, and the various portal tributaries injected with trypan blue solution; the animals were killed with ether soon after the injections, and the liver was removed for examination. Injections were, as a rule, made into the smallest veins that could be easily entered by a hypodermic needle; care was taken not to disturb the normal position of the viscera when the injections were being carried out. About thirty seconds was the average time taken to complete an intravenous injection, and only gentle pressure was employed. In some instances, the position of the animal on the operating table was altered in order to study the effect of posture. The experiments were repeated many times for each part of the portal vein that was under investigation. In all, about 100 experiments were performed.

Sites of Injection and Results.—In the following descriptions, the liver is regarded as being divided into right and left halves by a line passing from the fossa for the gallbladder to the entrance of the hepatic veins into the inferior vena cava. It was noted that in the canine liver this division does not correspond to the anatomic distribution of the two main branches of the portal vein.

Injection of Splenic Vein: A venule at the lower end of the hilum was selected for injection. Almost the whole of the left lobe of the liver was colored by the dye. It was observed that a limited area of the upper part of the extreme right

lobe of the liver constantly received a portion of the dye. The remainder of the right side of the liver retained its normal chestnut color, and the dye did not register itself in any part of it.

Stomach: The injection was made into a small vein on the lesser curvature. The whole of the left half of the liver was uniformly stained by the dye. The line of separation between the right and left lobes was sharply demarcated and thrown into striking contrast by the unilateral staining of the liver. No trace of dye was recognized in the right side (fig. 3).

Pancreas and Upper Part of Duodenum: An injection was made into the superior pancreaticoduodenal vein. The dye was constantly carried to the right side of the liver; the two lobes on the extreme right of the liver were deeply stained. A small amount was deposited in the right border of that lobe which adjoins the two more lateral lobes (fig. 4).

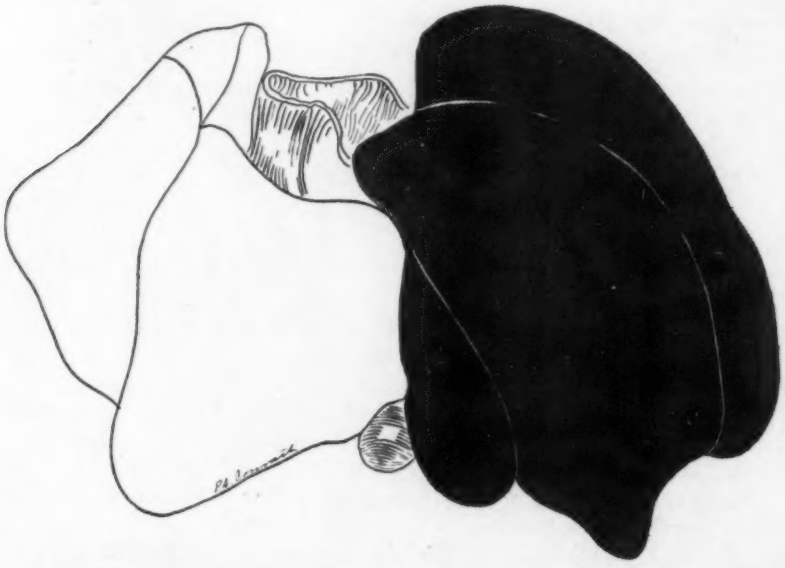

Fig. 3.—Dog's liver drawn from a specimen to illustrate the distribution of trypan blue after the injection into a gastric branch of the splenic vein. The black area denotes the part of the liver that was stained by the dye.

An injection was made also into the inferior pancreaticoduodenal vein. The dye was carried mainly to the right half of the liver, although a certain amount reached the left side. The lobe which lay immediately to the right of the gallbladder always showed deeper staining than any other part of the liver.

Jejunum: Dye was injected into a mesenteric vein at the highest part of the jejunum. The bulk of the dye registered itself in the two extreme right lobes of the liver. Scattered spots of coloration were seen occasionally in the left half of the liver but never more than a few (fig. 5).

Cecum and Appendix: A small vein at the root of the meso-appendix was selected for injection. The dye was transported to all parts of the liver. The large outer left lobe of the liver generally showed a greater deposition of dye than other parts, and was stained more deeply than the rest of the liver. In some dogs, the right half of the liver showed a greater amount of dye than the left.

Descending Colon and Rectum: The large vein in the mesentery of the left part of the colon was selected for injection. The dye was distributed throughout the liver. A greater amount of dye was evident in the left side, more especially in the large lobe on the extreme left side of the liver (fig. 6).

Fig. 4.—Distribution of dye after the injection of trypan blue into the upper pancreaticoduodenal vein of the dog. The darkly shaded area indicates the part of the liver to which the dye was carried.

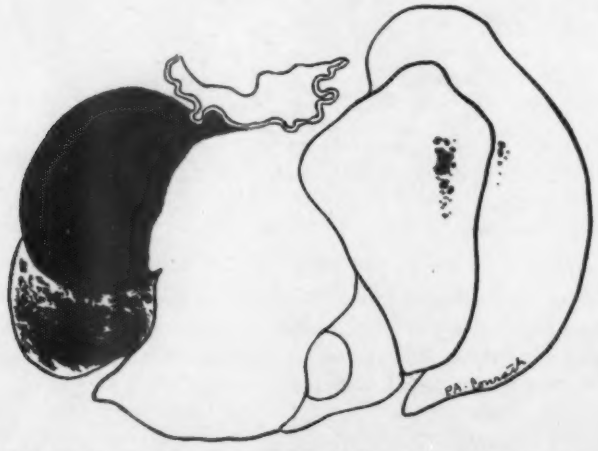

Fig. 5.—Distribution of dye after the injection of a mesenteric vein of the upper part of the jejunum of the dog.

Large Mesenteric Vein: The dye was injected at a point just distal to the entrance of the small mesenteric vein. The dye was deposited in the two lobes at the extreme right of the liver; a little was carried to the lobes of the left side.

The Effect of Posture.—Alteration of the position of the animal on the operating table, such as turning the trunk to one side or supporting the animal in the quadruped position while an injection was made, did not alter the character of the experimental observations.

The Disappearance of the Dye from the Liver.—In some instances, the behavior of the dye in the liver was observed before the animals were killed. It was noticed that when the dye had restricted itself to a particular portion of the liver there was only a slight tendency for diffusion to occur to a part beyond the initial site of localization. After from five to ten minutes, the dye commenced to disappear from that part of the liver in which it had first deposited itself. The liver as a whole assumed a slightly darker color throughout, but only to the same degree as the other organs of the body that, for a time, became tinted light blue when the dye reached the systemic circulation.

The Effect of Large Injections of Dye.—When the trypan blue was injected in a large quantity the liver was colored throughout, but the staining was not equal in all parts; for example, if a large volume of dye was injected into the splenic vein the whole liver became blue, but the staining was always more pronounced in the left side.

Fig. 6.—Distribution of dye after injection of a vein in the mesentery of the lower part of the colon.

VISUALIZATION OF THE CURRRENTS WITHIN THE PORTAL VEIN

Method.—The peritoneum was denuded from the surface of the portal vein and its larger tributaries near their termination. The experiments were carried out in a darkened room. A Cameron curved abdominal light that transilluminated the interior of the vessels was placed immediately behind the portal vein. The moving blood current was evident at once, when the light was placed in situ. Small dogs were used in these experiments so that effective transillumination of the portal vein could be obtained more readily. Trypan blue dissolved in blood serum was introduced into different branches of the portal system, and the transilluminated portal vein was kept under observation while the injections were being made. The injections were made slowly and with great gentleness.

A striking demonstration of sharply defined intraportal currents was afforded by this method, and the extreme rapidity of blood flow in the portal vein was made evident.

Sites of Injection and Results.—Splenic Vein. The injection was made near the hilum of the spleen. The dye-stained blood was seen to enter the left side of the portal vein obliquely. The dark stream kept strictly toward the left side of the

portal vein and preserved a narrow, ribbon-like course throughout the portal vein up to the hilum of the liver. No mixing with the adjacent blood in the portal vein was observed, and a cleancut linear division between the narrow splenic and the broad mesenteric streams was seen throughout the length of the portal vein. The injection was continued for several minutes, and at no time was there any alteration in the character of the current.

Injection of Jejunal Vein: A vein in the mesentery of the upper part of the jejunum was selected for injection. The stream was narrow and was sharply demarcated. It maintained an undeviated course along the right wall, and no mixing with red blood was seen to occur as it traveled to the hilum of the liver.

Small Mesenteric Vein: The large vein in the mesentery of the descending colon was selected for injection. A thin linear current of blood was visualized in the center of the ventral aspect of the portal vein. The current preserved an absolutely undeviated course throughout the length of the portal vein, and diffusion into other parts of the vessels did not occur.

COMMENT

We have found by the injection of trypan blue into tributaries of the portal vein of dogs that, with few exceptions, the dye is transported in the portal blood stream to fairly constant and definite areas in the liver, and that the site varies according to the place at which the dye is introduced peripherally. In our experiments, we found that when trypan blue was injected into the veins of the stomach and spleen it was conveyed by the blood to the left half of the dog's liver almost entirely. Dye injected into the veins of the upper part of the duodenum, the head of the pancreas and the jejunum was carried to the right lateral lobes almost exclusively. When the veins of the colon were injected, the dye was distributed to all parts of the liver, but more particularly to the large lobe of the left side. In those instances, in which the two colonic veins did not unite in a common mesenteric trunk but had a separate termination in the large mesenteric vein, it was found that injection of the right vessel resulted in a more liberal distribution of the dye in the right side of the liver.

Trypan blue stains the parenchyma of the liver and the coloration remains for some time. Once the dye is carried to a particular region of the liver, the staining remains sharply circumscribed. The terminal character of the lobar divisions of the branches of the right and left portal vein and the absence of interlobar and intralobar venous anastomoses are responsible for the absence of visible diffusion to other parts of the liver. The temporary staining of the liver does not signify that the blood which conveyed the dye has itself remained confined in a demarcated area of the liver for so long a period, as the dye is almost immediately recognizable in the systemic circulation after intraportal injection; furthermore, the volume of flow of portal blood through the liver is rapid; in the dog, 60 cc. of blood per hundred grams of liver in a minute.

It may be inferred from these facts that possibly blood that has been conveyed from the periphery of the portal system to a particular section of the liver is utilized in that specific area without contact with blood from other parts of the portal tree. It is of interest to note that, according to our experimental observations, the blood which is carried to the left lobe of the liver is drained principally from those abdominal organs that are not strictly engaged with the digestion or absorption of food, namely, the spleen, the stomach and the greater part of the colon; whereas, the blood collected from those parts of the alimentary tract where the products of digestion are absorbed is carried to the right side of the liver and, in the dog, often to a special and constant site in that lobe. For example, the blood from the upper pancreaticoduodenal veins is conveyed to the two lateral lobes of the right side of the liver.

In brief, it would seem from our evidence that there are areas in the liver which are especially supplied with blood from specific parts of the portal system, and that the blood from the different parts of the portal system does not become mixed in the liver nor, as we shall indicate, in the portal vein itself.

An explanation for the selective distribution of the portal blood in the liver is afforded by the visual demonstration of stream lines in the transilluminated portal vein after the injection of dyes. These currents serve to transport blood from the periphery of the portal system to the liver in such a manner that little or no intermingling of the individual blood streams occurs in the main portal channel. It would seem from our experiments that there are at least three main blood currents in the portal vein. These currents are, in our opinion, derived from three sources, namely, the splenic vein, the large mesenteric vein and its branches and the small mesenteric vein. In our observations, three sets of currents were initiated and maintained in the portal vein by these three large vessels.

Maintenance of individuality of different currents in the portal vein finds a counterpart in at least one other human circulatory system. In the fetal heart, blood from the superior and inferior vena cava does not mix freely in the right auricle. Likewise, the individual currents in the portal vein may be likened, in part, to the union of two rivers, for example, the Rhone and the River Avre, one clear and the other muddy, that retain their identity for a considerable distance after their union in a common stream-bed. This phenomenon of separate streams in a moving body of water is well known to hydraulic engineers and is usually discussed under the name of "stream lines." The anatomic disposition of the splenic, large mesenteric and small mesenteric veins would seem to be favorably adapted to the ejection of blood into the main portal channel in such a direction that the

individual streams could remain separate. These anatomic advantages are probably further aided by the known differences in the rate of flow in the veins, their relative tonicity and differences in viscosity of the blood they contain.

SUMMARY AND CONCLUSIONS

1. The distribution of the portal blood in the liver of the dog was determined by the injection of a dye.

2. Blood from the spleen, the stomach and a greater part of the colon goes to the left lobe of the liver; whereas blood from the duodenum, the head of the pancreas and the upper part of the jejunum goes mainly to the right lobe of the liver.

3. A satisfactory method of visualizing experimentally the individual currents of blood in the portal vein is presented.

4. There are at least three distinct and separate currents in the portal vein.

5. The anatomy of the portal vein and the intrahepatic distribution of its blood in the dog were studied and the results have been presented.

THE RÔLE OF THE CIRCULATION IN THE HEALING OF FRACTURES

A REVIEW

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Normal Blood Supply of Bone.
Effect of a Fracture on the Adjacent Arterial Circulation.
Effect of Cutting the Veins and Lymphatics at the Site of Fracture.
Incompressibility of Muscle Tissue and the Effect of This on the Circulation of Blood to a Fracture Area.
Relation of the New Bone Trabeculae to Blood Vessels.
The Laying Down of Lime Salts.
Development of Fibrous Tissue.
Summary.
Conclusions.

In 1863, Hamilton [1] estimated the incidence of nonunion of fractures to be one in 500 cases. Scudder and Hey-Groves,[2] in 1927, placed it, respectively, at from 2 to 3 and from 3 to 5 per cent. If it is true, but this is doubtful, that there is a larger percentage of delayed unions and nonunion of fractures today than formerly, it may easily be accounted for by the greater traumatisms sustained today and by the more exacting standards under which the surgeon works, but whether it is true or not, the fact still remains that a better conception of all the vital processes concerned in bone repair will result in diminished morbidity.

In this article are presented some salient facts relative to the circulation and its possible relation to the repair of a fracture.

The repair of a fracture is by a group of interrelated processes by no means well understood. These processes are: (1) the establishment of the circulation in preparation for the definite construction of bone; (2) dissolution, partial or complete, of the old bone and the laying down of lime salts to form new bone, which, architecturally, is not the same as the old, and (3) rearrangement of the new bone to carry the particular stress to which that section of bone is subjected.[3] While these processes occur in the order given, they overlap so that two or even all may be going on at the same time. Throughout all processes of bone repair, the circulation is of primal importance, and by this is meant, not only the arterial circulation, but that of the veins and lymphatics, for without the elimination of the waste products, repair will be impossible.

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1. Hamilton, F. H.: A Practical Treatise on Fractures and Dislocations, Philadelphia, Blanchard and Lea, 1863, p. 61.

2. Bancroft, F. W., and Scudder, C. L.: The Treatment of Fractures, ed. 10, Philadelphia, W. B. Saunders Company, 1926, p. 949.

3. Koch, John C.: The Laws of Bone Architecture, Am. J. Anat. **21**:177 (March) 1917.

NORMAL BLOOD SUPPLY OF BONE

The outer layers of the long bones are nourished by the periosteum; the inner layer, by the medulla. Blood vessels run longitudinally through the bone in a series of canals called the haversian canals, around which the bony lamellae are grouped. Another series of vessels passes from the periosteum into the interior of the bone through minute orifices. The medullary or nutrient arteries enter the bone through the periosteum and, traversing the cortex at a sharp angle, enter the medullary cavity, the length of the artery in the cortex being relatively short. The epiphysis is supplied from vessels which anastomose around the joints from three sets of vessels: the diaphyseal, the metaphyseal and the epiphyseal; thus epiphyseal vessels communicate with those in the metaphysis,[4] and as the ramifications of the medullary or nutrient artery connect with the vessels in both the compact and the cancellous bone, anastomoses exist among all the arterial vessels in a bone. Johnson[5] maintains that the metaphyseal arteries are second in importance to the nutrient arteries in nourishing the medulla; the inner half of the cortex receives its nutrition from the medulla, and the outer half of the cortex is nourished by the periosteal vessels.

The periosteal blood vessels subdivide rapidly before entering the cortical bone. As the blood vessels enter the haversian and Volkmann's canals, they are surrounded by delicate sheaths of areolar tissue; through continuity with the endosteal arcade, this areolar tissue is attached to the wall of the canal, and this gives support to the more or less centrally located vessels.[6]

The veins and lymphatics are probably no less important to the healing of fractures than are the arteries. The veins emerge from the bone in three places: (*a*) one or two large veins accompany the medullary (or nutrient) artery; (*b*) numerous large and small veins emerge at the articular extremities, and (*c*) many small veins pass out from the compact substance.[7] Lymphatic vessels have been traced by Cruikshank into the substance of the bone, and Klein describes them as running in the haversian canals.[7] A point that one should keep in mind is that when bone is divided, its vessels do not contract into the canals in which they are contained.[7]

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4. Boyd, W. M.: Surgical Anatomy, Philadelphia, W. B. Saunders Company, 1925.

5. Johnson, R. W., Jr.: A Physiological Study of the Blood Supply of the Diaphysis, J. Bone & Joint Surg. **9**:153 (Jan.) 1927.

6. Blaisdell, F. E., and Cowan, J. F.: Healing of Simple Fractures, Arch. Surg. **12**:619 (March) 1926.

7. Gray, Henry; and Lewis, W. H.: Anatomy of the Human Body, Philadelphia, Lea & Febiger, ed. 21, 1924, p. 87.

EFFECT OF A FRACTURE ON THE ADJACENT ARTERIAL CIRCULATION

It may be accepted as axiomatic that repair of a fracture depends on the establishment of adequate channels of blood circulation to the injured area. Kolodny has shown by injected specimens [8] that following a fracture there is increase in the diameter and in the tortuosity of all the arterial vessels in the section of the limb adjacent to the fracture.

Coincident with the occurrence of a fracture of a long bone there is damage to the normal blood supply. The longitudinal vessels are torn or cut by the shearing action of fragments, and the periosteal vessels are torn where the periosteum is separated from the cortex of the bone by avulsion. Bleeding immediately occurs from the injured vessels. In due time, a clot is formed about the fragments; lineal projections of this clot extend into the lumen of the arterial vessels until they reach the nearest patent collateral, and thus thrombi are formed in the vertical trabecular spaces of the bone for a varying distance below and above the actual line of fracture.

The clot in the longitudinal bone channels, i.e., the thrombi and that between the fragments, must be spoken of separately. The former no doubt will resolve by the same processes as will a thrombus of soft parts, where granulation tissue gradually replaces the clot and develops terminally into fibrous tissue with obliteration of the vessel, waste products being carried off by the leukocytes in the venous or lymphatic stream.[9] With all the arterial longitudinal vessels crossing the site of the fracture blocked by thrombi and in progress of obliteration, any blood supply from them to the seat of fracture is impossible. The clot between the fragments, however, is gradually replaced by granulation tissue,[10] which results in a close network of thin-walled vessels. These vessels become thicker by the growth of adventitial cells, some of the connective tissue and others of the plasma cell group; thus fibrous tissue develops progressively. Obviously, at this time there is little help in repair given by the arterial vessels of the cortex, which are plugged by thrombi. The granulation tissue starts from buds from functioning vessels. These may be from the medullary cavity, but they are mostly from the area surrounding the fracture,[11] where the greatest activity,

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8. Kolodny, A.: The Periosteal Blood Supply and Healing of Fractures, J. Bone & Joint Surg. **5**:698 (Oct.) 1923.

9. Keen, W. W., and DaCosta, J. C.: Surgery, Its Principles and Practice, Philadelphia, W. B. Saunders Company, 1920, vol. 1, p. 418; vol. 2, p. 104; vol. 5, p. 165.

10. Blaisdell and Cowan (footnote 6). Bancroft and Scudder (footnote 2, p. 927). Eising, E. H.: Remarks of the Physiology of Bone in Relation to Bone Diseases, Am. J. Surg. **3**:368, 1927.

11. Bancroft and Scudder (footnote 2, p. 927).

the most active circulation and the greater area exist. At about this stage, then, the clot is reduced in volume and is undergoing organization. It consists essentially of a moist, gluey weld of extraordinary abhesiveness. The new growth of vessels is horizontally [12] inward and no doubt horizontally outward from the medulla, providing, of course, in the case of the latter, that the injury has not entailed the destruction of the blood supply of the medullary cavity. Kolodny believes that "the endosteum is not able to participate in the callus formation until the intraossol blood circulation recovers by formation of an anastomosis between the central portion of the nutrient artery and the metaphyseal blood vessels." [8]

EFFECT OF CUTTING THE VEINS AND LYMPHATICS AT THE SITE OF FRACTURE

The same shearing force that cut the arteries also must cut the veins and lymphatics, and as reposition of the fragments is practically never so accurately made as to bring the cut ends of the vessels into venous alinement, the flow of venous blood and lymph will not be reestablished across the fracture in the vessels except by the probable development of new vessels; however, this will not interrupt the carrying capacity of the veins and lymphatics, as will appear. While the cut arterial vessels are thrombosed to their nearest functioning collateral, the veins seldom are. The venous circulation has for its motivation: (*a*) the propulsive power of the heart, (*b*) the compressive effect of muscle action and (*c*) the suction from the thorax and the heart itself.[13] The propulsive power of the heart on the venous column, slight in any case, is of course nonoperative in the cut vessels, but the suction from the thorax and heart and the presure of surrounding parts are still present. The valves in the veins and lymphatics prevent any flow of venous blood or lymph in any direction except toward the heart. Smaller veins (of less than half an inch, such as lie in bone) do not have valves.[7]

INCOMPRESSIBILITY OF MUSCLE TISSUE AND THE PROBABLE EFFECT OF THIS ON THE CIRCULATION OF BLOOD TO A FRACTURE AREA

There can be no doubt concerning the relative incompressibility of muscle tissue. Only by great pressures (those tremendously in excess of any existent in the body) can any compressibility be demonstrated.

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12. Wieder, Henry S.: Regeneration of Bone, Univ. Penn. M. Bull., 1907, p. 33.

13. Tigerstedt, R.: A Testbook of Physiology, ed. 3, New York, D. Appleton & Co., 1906, p. 19. Starling, E. H.: Principles of Human Physiology, ed. 4, Philadelphia, Lea & Febiger, 1926, p. 872.

Henderson and Brink [14] demonstrated this in 1908 by carefully conducted physical experiments. While muscle changes its shape in contracting and expanding, it does not change its volume. Since muscle is a colloidal solution, albeit retained in its form by its sheath or its other fibrous interstitial structures, it will have the physical characteristics of a fluid. In the case of a fracture of a long bone, pressures are created against and in the muscle: (1) by displacement by hemorrhage, (2) by hemorrhage into its own interstitial structure from the trauma which caused the fracture and (3) by infiltration of inflammatory products around and into the muscle tissue. This increase in volume is apparent as swelling and is not in itself harmful or prejudicial to repair of a fracture; however, if such swelling is prevented, a series of maladjustments in pressures ensue which are deleterious not only to the circulation to the fracture, but to the muscle tissue itself. As an example of the latter, ischemic paralysis may be mentioned. In this article, only the effects of pressure on the circulation of the fracture are considered. It has been shown how pressure in and about muscles is created; this pressure may be resisted by heavy layers of fascia and by tightly applied splints, bandages, adhesive plaster or casts. If resistance to expansion (swelling) occurs, its effect will be to diminish or occlude the lumen of the arterial blood vessels and thus prevent an adequate supply of blood from reaching the interior of the callus. I believe that, regardless of the exact manner in which mineral salts are laid down, they are carried by the arterial system to the region in process of repair. If this belief is correct, interference with an adequate supply of blood must have a deleterious effect. It will be shown subsequently that granulation tissue is converted into fibrous tissue, which, by its inherent power of contraction, eliminates the very vessels by which it was formed. If, however, sufficient bone tissue has been laid down while the new vessels were still patent, they will make a more or less permanent connection with the general circulation and persist through the period of their need. Hence the great importance of an effort to prevent considerable hemorrhage and inflammatory reaction even during the early days of a fracture is evident, or if this should occur, the immediate provision for the additional expansion by a readjustment of any circumferential dressing whether it is a bandage, cast or adhesive plaster.

While a generalized pressure of surrounding tissues may occlude the arterial part of the circulation to a fracture, it has the opposite effect on the veins and incidently also on the lymphatics. These vessels are provided with valves which, not allowing a backward flow, cause the thin contained fluid (blood or lymph) under pressure to move in only one

14. Henderson, L. J., and Brink, F. N.: The Compressibilities of Gelatine and Solutions of Muscle, Am. J. Physiol. **21**:248 (Feb.) 1908.

direction; this, of course, is toward the heart and away from the fracture. It may also be remembered that the venous and the lymph streams are subject to a suction action by the thorax and heart. It would seem not unreasonable to presume that these factors acting together might create a negative pressure in the venous lymphatic channels in the bone, sufficient to carry off waste products, even if of little magnitude. There is plenty of evidence that in the tearing down process of the old bone, the products are carried away by the old vessels, that is, those running in a vertical direction. As evidence of this, there are the clear vertical spaces (erosion) shown in the old bone; one sees this in the cuts of sagittal sections of repairing bone, and it has been shown particularly in many articles on the subject of bone repair, for example, by Johnson,[5] by Todd and Iler,[15] by Wieder [12] and by others. The eburnation so regularly (though not invariably) seen in cases of nonunion would seem to be a point in favor of believing that in these cases resorption of bone salts and other substances is through the veins and lymphatics.

RELATION OF NEW BONE TRABECULAE TO BLOOD VESSELS

The arrangement of the new trabeculae at the zone of fracture is at right angles to the cortex rather than parallel to it as in normal bone. This is presumably due to the fact that, in general, the new blood vessels growing into the original granulation tissue were derived from the surrounding soft tissues outside the bone and from the medullary cavity. The realinement of the vessels of the cortex is not complete for a year or more after the injury.[3] Delafield and Prudden, speaking of the ossification of osteophytes, said:

> The blood vessels from which the pathological bone develops grow out of old vessels, and as in the formation of granulation tissue, are irregularly arranged and are subject to a variety of nutritive conditions, so that new bone is usually formed, not in a series of definite lamellae but in a series of irregular spicules or masses.[16]

Blaisdell and Cowan[6] have stated: "The manner of branching and the position of the blood vessels determine the form, position and manner of continuity of the early trabeculae." The progressive change in the direction of the trabeculae from the horizontal to the vertical is probably due to gradual changes in the anastomotic formation of the blood vessels (particularly of the arteries) by virtue of which they (the trabeculae) tend to conform to the direction of the vessels of the bone as a whole.

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15. Todd, T. W., and Iler, D. H.: The Phenomena of Early Stages of Bone Repair, Ann. Surg. **86**:715, 1927.

16. Delafield, Francis, and Prudden, T. M.: A Textbook of Pathology, ed. 12, New York, William Wood & Company, 1923, p. 1059.

THE LAYING DOWN OF LIME SALTS

The deposition of mineral salts to form hard bone involves a different group of phenomena from those under consideration in this article. The theory that the osteoclasts and osteoblasts have the particular property of tearing down and building up bone does not have the universal recognition today that it did ten years ago,[17] although many writers still accept it. It has been shown that the formation of new blood vessels is mostly horizontal, and that the new trabeculae are arranged in the same direction. Ely [18] and later Blaisdell and Cowan [6] found, in their experiments on animals, that new bone was first laid down in the angle formed by the bone and the raised periosteum. This is the place where one would expect that anastomosis would first take place between the periosteal circulation and that in the cortex of the bone. It is also the place where the effect of motion between the fragments would be minimized as being farthest from the place of shearing action.

DEVELOPMENT OF FIBROUS TISSUE

The result of the failure of bony union, with all it implies concerning the impossibility of reconstruction of the architecture of the normal bone, is a fibrous union between the fragments. The fact has been mentioned that granulation tissue replaces the blood clot following a fracture. Granulation tissue develops ordinarily into fibrous or scar tissue. In a small wound, this development will take place quickly and almost at the same time throughout the small area; but in a large wound, the process will be progressive and farthest advanced at the place where the granulation tissue started to bud or form. In the case of most large wounds, this will be at the periphery so that the progressive healing is from without inward, or as J. Renfrew White [19] put it, centripetally. In the case of a fracture, there are two bases from which the granulation tissue may start to form; these are the outer periphery of the clot, with which the periosteum is usually associated, and the medullary region, providing, in the case of the latter that the blood supply to this region has not been stopped. With a proper clot and no interruption to its vascularization and the laying down of mineral salts, bone healing will be carried out. When interruption of the continuity of the vessel system takes place,

17. Eising (footnote 10, third reference). Arey, L. B.: The Origin, Growth and Fate of Osteoclasts and Their Relation to Bone Absorption, Am. J. Anat. **26**: 315, 1919-1920. Watt, J. C.: Deposition of Calcium Salts in Areas of Calcification, Arch. Surg. **10**:990 (May) 1925. Bancroft, F. W.: Bone Repair Following Injury and Infection, Arch. Surg. **5**:646 (Nov.) 1922.

18. Ely, L. W.: Experimental Study of the Healing of Fractures, Arch. Surg. **5**:527 (Nov.) 1922.

19. White, J. R.: Chronic Traumatic Osteomyelitis, New York, Paul B. Hoeber, 1919, p. 28.

however, the normal bone repair will be interrupted or fail completely, and the characteristic fibrous union will occur. It has been said that the granulation tissue leading to vascularization starts on the periphery of the clot and possibly in the medullary cavity. From these places it is progressively inward and outward. The horizontal series of new arterial vessels may have its continuity interrupted by cutting from the shearing action of the two fragments if moved, and in practice this is done by too frequent or not sufficiently gentle, firm handling in dressing, or it may occur from lack of proper support by the splint, the adjustment of which may permit hingelike motion between the fragments. I cannot see any reason for the belief held by some that "a little motion is good for a fracture"; by this is not meant the gentle contractions of muscle, as, for instance, the motion by the fingers in a forearm fracture, the effect of which is to accelerate the supply of blood to the fracture and to facilitate the elimination of waste by the veins and lymphatics.

If the continuity of the vessels is broken, it will not stop the normai histologic progress of granulation into fibrous or bone tissue at the periphery of the healing area, but only in the newly injured area between the fragments; therefore, the development of granulation into fibrous and perhaps even bone tissue will continue here (at the periphery), while the central area must have its vessel system repaired before it can again take part in the normal process of repair, namely, vessel formation, laying down of lime salts, etc. The inevitable tendency of granulation tissue is to form fibrous tissue, which contracts and deprives this central area of its blood supply; without this, it cannot make the necessary repair of its continuity. No doubt, occasional damage to the new vessels between the fragments is repaired without prejudice to the final result, but if such damage is repeated too often, it may result in deprivation of blood supply to this area through the gradual occlusion of the peripheral vessels of the callus by the formation of fibrous tissue.

SUMMARY

The deposition of lime salts incident to the repair of a fracture is dependent on an adequate circulation in the callus.

Following a fracture, a new set of arterial vessels transverse to the long axis of the (long) bone is developed. These vessels arise by a process of budding, starting, for the most part, at the periphery of the injured area; it must be admitted, however, that in the absence of obstruction to the medullary vessels, such budding may also arise from them. An anastomotic arterial system is thus established, and through its medium the trabeculae are laid down, at first transverse to the long axis of the (long) bone but later obliquely and eventually vertically, as is normal, this being brought about by the ever changing anastomoses of the vessels. The lime salts are removed and replaced according to the

exigencies of the process. Repeated injury to this delicate anastomotic network through motion of the fragments makes necessary repair by the formation of new vessels; this involves retardation in the interior of the callus while at the periphery granulation tissue is progressing to fibrous tissue; the latter contracts in due time and thus reduces the flow of blood to the interior of the callus. As a result, there is delayed or fibrous union.

Since muscle tissue is incompressible (by ordinary pressures), the addition of the volume of the hemorrhage and inflammatory exudate may reduce the lumen of the arterial supply either directly or indirectly by the transmission of pressures throughout adjacent muscles in accord with well known laws of hydraulics.[20] This is particularly likely to occur in the presence of resistance to expansion or swelling from fascial planes and circular bandages or casts.

The circulation through the veins and the lymphatics is of equal importance to that in the arterial system, because through their medium the old structure is torn down or resorbed, thus permitting the growth of new vessels to form and establish anastomoses which are essential to bone repair. Interruption to the flow of venous blood and lymphatic fluid by the same pressure that may retard the arterial supply does not occur because a generalized pressure on the veins and lymphatics forces their contained fluid toward the heart.

CONCLUSIONS

Whether or not there are any systemic causes for delayed union and nonunion of fractures, there would seem to be ample reason to believe that the consistent use of methods of treatment of fractures that require (1) good apposition, (2) adequate provision for expansion of surrounding structures and (3) freedom from any shearing or hinge motion between the fragments will be rewarded by a much lessened morbidity percentage. Good apposition is, of course, routine and needs no word of defense. Ample provision for expansion is not always provided in the cast, splint or other device used. Too much emphasis cannot be laid on the importance of splitting or bivalving the cast or in maintaining a constant watchfulness over circumferential bandages that they may never be too tight. A bandage may be correct at the time of its application, but over night, from the restlessness of the patient or from his having had the injured part in a dependent position, the limb may have swollen until the bandages are tight.

Much can be done at the first dressing to prevent excessive hemorrhage and inflammatory reaction. Usually at this time one is dealing

20. King, H. W., and Wisler, C. O.: Hydraulics, New York, John Wiley & Sons, 1922, p. 7.

with the break before the muscles have become spastic; there is only moderate swelling, and replacement of the fragments may be possible. The part should be placed in the position of election. Thus a fracture of the elbow if seen shortly after its occurrence may be placed in acute flexion with little danger of excessive swelling subsequently, and in fractures of the lower leg, the foot may be turned in. It is unnecessary and contributory to excessive swelling for one to endeavor to make an exact diagnosis by manipulation; roentgen-ray examination should follow the first dressing as soon as possible and readjustment of any deformity shown may then be made as soon as convenient, after which the roentgen ray should be employed to verify the position of fragments.

The fracture appliance, of whatever type, should not permit of any motion of the bone at the site of the fracture. The old rule to splint the proximal and distal joints is a good one.

Fractures should not be dressed frequently, unless the cause is imperative, and in dressing should be handled firmly and gently so that there is no motion between the fragments.

Open operations should be so planned and conducted as to minimize the damage to the surrounding circulation. The use of a tourniquet is not advisable, since with it, one is not cognizant of the amount of damage being inflicted on the circulation. Fibrous tissue about the fragments should be removed and likewise eburnated bone which is too compact to permit formation of new vessels with the consequent anastomotic phenomena so important in repair of fractures.

STUDIES IN INTESTINAL OBSTRUCTION

IV. STRANGULATION OBSTRUCTION: A COMPARISON OF THE TOXICITY OF THE INTESTINE AND OTHER TISSUES AUTOLYZED IN VIVO AND IN VITRO *

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MINNEAPOLIS

In both the patient and the experimental animal, strangulating types of obstruction are the most serious. In addition to the occlusion of the bowel in strangulation obstruction a segment of intestine is deprived of its blood supply. The poorer prognosis and quicker fatal issue in such instances are due to the injury of the strangulated loop.

A study of strangulation obstruction, therefore, resolves itself into an inquiry into the autolysis of injured segments of intestine. It has been demonstrated in another study [1] that segments of intestine deprived of their blood supply undergo a rapid autolysis, with the liberation of toxic substances. In a few dogs in which strangulation obstructions had been established and the strangulating mechanism released a few hours later, death occurred, apparently caused by the absorption of toxic bodies from the strangulated segment. Autolysis of the segment to which the blood supply had been interrupted, rather than absorption from the lumen of such a segment, appeared to be responsible for the fall in blood pressure and the lethal issue observed in such animals on releasing the constriction of the strangulated segment.

In this investigation, comparisons have been made of the toxicity of various tissues when deprived of their blood supply. A study of the nonprotein nitrogen of the blood and urine has also been made in a number of these animals to note whether the autolysis of devitalized tissue would provoke the same alterations in the chemistry of the blood and urine as are observed in simple occlusion of the upper part of the intestine. An effort has also been made to ascertain whether the subcutaneous administration of sodium chloride to animals in the peritoneal cavities of which tissues were allowed to autolyze afforded the animal any protection.

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* From the Department of Surgery of the University of Minnesota.

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1. Wangensteen, O. H., and Loucks, M.: Studies in Intestinal Obstruction; II. The Absorption of Histamine from the Obstructed Intestine, Arch. Surg. **16**: 1089 (May) 1928.

METHOD

All surgical procedures were performed under ether anesthesia and aseptic technic. Blood was obtained from the jugular vein or the vein on the outer aspect of the hind leg of the dog. The blood urea was determined according to the method of Van Slyke and Cullen[2] and the blood chlorides after the method described by Gettler.[3] A number of the animals were kept in metabolism cages. The urine was collected and the nonprotein nitrogen determined by the micro-Kjeldahl method of Folin and Denis.[4] When sodium chloride was given, it was administered subcutaneously with a 50 cc. syringe; 400 cc. of a 2 per cent sodium chloride solution being given once daily. Segments of small intestine about a foot or a little more in length, weighing from 46 to 75 Gm., were removed from one dog and placed in the peritoneal cavity of another. In four instances, the loop was irrigated successively with water, alcohol, ether and water, until the contents were returned clear. About 2 liters of fluid were used in the irrigation of each loop. A large needle was inserted in one end, and the other end was left open. Irrigation was performed with a 50 cc. syringe. A few inches of both ends of the segment of intestine, where the irrigation was less effective, were cut off before the remainder of the segment was deposited in the peritoneal cavity of the other dog. In the other eight instances, following this irrigation, the bowel was slit on its antimesenteric border and the mucous surface painted with phenol in certain areas in order to destroy the mucous membrane. The destroyed mucosa was then scraped off with a knife. In a few instances, a major part of the mucosa was stripped off in this manner leaving only islands here and there.

The spleen was detached from its blood supply and dropped into the free peritoneal cavity in a number of animals. The same procedure was carried out with pieces of liver. The pancreas excised from other dogs was placed in the peritoneal cavity in two animals, and in a few animals kidneys excised from other dogs were used in the same way.

The twelve dogs from which segments of intestine were removed recovered following the establishment of the continuity of the intestinal tract by lateral anastomosis. Death occurred in eleven of the twelve animals into the peritoneal cavities of which the twelve segments of intestine were deposited. One dog recovered; in this animal, a good portion of the mucosa had been removed. Another animal lived seven days. Five dogs survived the procedure two days. The others died on the day following operation. One of the latter died in convulsions, a mode of death not uncommon in the rats injected intraperitoneally with the intestinal contents of either a dog or a rabbit. Two of the dogs that were given salt solution subcutaneously died the day following the placing of the intestinal loop into their peritoneal cavities. The heaviest segment of intestine weighed 75 Gm., the lightest 46 Gm., the average

2. Van Slyke, D. D., and Cullen, G. E.: A Permanent Preparation of Urease and Its Use for Rapid and Accurate Determination of Urea, J. A. M. A. **62**:1558 (May) 1914.

3. Gettler, A. O.: A Method for the Determination of Death by Drowning, J. A. M. A. **77**:1650 (Nov.) 1921.

4. Folin, O., and Denis, W.: Nitrogen Determinations by Direct Nesslerization; I. Total Nitrogen in Urine, J. Biol. Chem. **25**:473, 1916.

weight being 63.5 Gm. The average weight of the dogs into the peritoneal cavities of which loops of bowel were placed was 37 pounds (16.8 Kg.). The dog that recovered weighed 25 pounds (11.3 Kg.) and the segment of intestine, 52 Gm.

The four dogs into the peritoneal cavities of which pieces of liver were placed died. In one animal, the small lobe of the liver near the gallbladder was detached and dropped into the peritoneal cavity; at autopsy, there was no indication that bleeding had occurred from the liver. In the other animals, the pieces of liver used were obtained under sterile precautions from normal dogs killed during the course of another experiment. Three of these dogs weighed 35 pounds (15.9 Kg.) and the other 25 pounds. The weight of the segments of liver used averaged 56 Gm. One animal into the peritoneal cavity of which 35 Gm. of liver was placed, lived five days. The other three animals survived a little less than twenty-four hours. Three of the four dogs were given saline solution subcutaneously.

Of twelve dogs in which the animal's own spleen was detached from its source of blood supply and dropped into the peritoneal cavity, eight died and four recovered. The weights were not obtained in some of these animals, but there did not appear to be any uniformity between the size of the spleen, the weight of the dog and recovery. One animal weighing 26 pounds (11.8 Kg.) recovered following detachment of a spleen that weighed 82 Gm. Another dog that weighed 21 pounds (9.5 Kg.) died two days following the detachment of its spleen, the weight of which was only 22 Gm. Six dogs were given salt solution subcutaneously. One dog was given water subcutaneously. Postoperative treatment was not given to the remaining five animals. Of the four dogs that recovered, all were given saline subcutaneously. The average length of survival following the operative procedure on the other dogs was three and a half days.

A kidney removed from other healthy dogs was placed in the peritoneal cavity of two dogs, each weighing 25 pounds. The weights of the kidneys used were 34 and 29 Gm., respectively. One of these dogs died eighteen days later from a suppurating abdominal wound; the other dog made a permanent recovery. Two kidneys were placed in the peritoneal cavity of a third dog. The combined weight of the two kidneys was 60 Gm. This dog also survived the procedure. The employment of tissue obtained from another animal did not seem to be of any special handicap in recovery.

In order to establish better quantitative comparisons of the toxicity of various tissues in the process of autolysis, pieces of tissue weighing from 30 to 60 Gm. were removed under sterile precautions from two dogs killed with ether. The pieces were placed in sterile bottles with wide mouths containing about an equal weight of physiologic sodium

chloride, and the bottles were then placed in the incubator. At twenty-four, forty-eight, seventy-two and ninety-six hours following the incubation, a few cubic centimeters of fluid was aspirated from the bottles and injected intraperitoneally into rats. A sufficiently large number of

TABLE 1.—*Results of Injection of Autolyzed Tissue After Sterilization*

Tissue	Hours Incubated	Amount Injected, Cc.	Result	Remarks
Kidney	24	18	Recovery	Mildly ill
	48	12	.....	Died 2 days later
	48	10	Recovery	Fairly ill
	72	5	Recovery	Stuporous, but recovered
	96	10	Died	Mildly ill after injection; died 2 days later
	4 months	4	Died	Convulsions 10 minutes after injection
Spleen	24	15	Recovery	Mildly ill
	48	12	Recovery	Mildly ill
	48	10	Recovery	Mildly ill
	72	5	Recovery	Stuporous, but recovered
	96	10	Died	In convulsions 5 minutes after injection: convulsive seizures for 45 minutes; died next day
	4 months	4	Died	Very ill; died during night
Liver	24	15	Died	Moderately ill; died during night
	48	12	Died	Moderately ill; died during night
	48	10	Recovery	Fairly ill
	72	5	Died	Stuporous; found dead in a.m.
	96	10	Died	Not as ill as other animals injected with 4 day tissue culture; died 2 days later
	4 months	4	Died	Stuporous; died 12 minutes later
Pancreas	24	15	Died	Dead 20 minutes after injection
	48	12	Died	Died during night
	48	10	Died	Died during night
	72	4	Recovery	Stuporous following injection
	96	10	Died	Moderately ill after injection; died 2 days later
	4 months	4	Died	Convulsions; dead in 5 minutes
Duodenum	24	17	Died	Dead 30 minutes after injection
	48	12	Died	Died during night
	48	10	Died	Died during night
	72	5	Died	In convulsions 3 minutes after injection; dead 2 minutes later
	96	10	Died	Convulsive seizures; dead a few minutes after injection
	4 months	4	Died	Very ill; dead in 30 minutes
Jejunum	24	15	Died	Dead 7 minutes after injection
	48	12	Died	Very ill; died a few hours after injection
	48	10	Died	Very ill; died a few hours after injection
	72	5	Died	Stuporous; died during night
	96	10	Died	Convulsive seizures; dead 10 minutes after injection
	4 months	4	Died	Convulsions; died in 10 minutes
Stomach	24	18	Died	Dead 7 minutes after injection
	48	12	Died	Very ill; dead a few hours after injection
	48	10	Died	Very ill; dead a few hours after injection
	72	5	Recovery	Stuporous, but recovered
	96	10	Died	Convulsive seizures; dead 4 minutes after injection
	4 months	4	Died	Very ill; died in 20 minutes
Muscle	24	15	Recovery	Mildly ill
	48	12	Died	Died during night
	48	10	Died	Died during night
	72	5	Recovery	Stuporous, but recovered
	96	10	Died	Lethargic and stuporous; died during night
	4 months	4	Died	Convulsions; dead in 12 minutes

bottles were prepared so that each day's injection was made from a separate bottle. Injections of tissues autolyzed in this manner were made with liver, spleen, pancreas, muscle, kidney, stomach and intestine.

In the incubator, tissues would undergo about the same qualitative process of autolysis as the same tissue deprived of its blood supply when

placed in the peritoneal cavity. In the body cavity the rate of autolysis would be somewhat increased because of the simultaneous action of living tissue in aiding the disintegration of the dead tissue. Table 1 shows the results of such injections, the results checking closely with those obtained previously when the same tissues were placed in the peritoneal cavity.

COMMENT

When the loops of the bowel removed from one dog and made bacteria-free by mechanical cleansing and irrigation with antiseptic solutions are placed in the peritoneal cavity of another animal, death quickly supervenes as a consequence of autolysis of the segment of intestine deprived of its blood supply. Such segments of bowel, averaging 63.5 Gm. in weight, were placed in the peritoneal cavities of twelve dogs. Only one recovered; in this animal, a considerable portion of the mucosa had been scraped off. In two animals, a portion of the mucosa was removed from the segment of bowel, and each of the dogs into the peritoneal cavities of which the prepared segments of bowel were placed was given 400 cc. of a 2 per cent sodium chloride solution subcutaneously. Both dogs died the next day.

Four dogs died after pieces of liver averaging 56 Gm. were placed in the peritoneal cavities. A salt solution was given subcutaneously to three of these animals, but without avail.

Kidney tissue removed from other dogs was placed in the peritoneal cavity of three dogs. One death occurred eighteen days later from a suppurating abdominal wound; the other two animals recovered. Saline solution was not given to any of these animals.

Death occurred in eight of the twelve dogs in which the spleen was detached from its source of blood supply and dropped into the peritoneal cavity. Six of the twelve dogs were given salt solution subcutaneously; the four dogs that recovered belonged to this group. The five dogs to which salt solution was not given subcutaneously died, as did the animal given water subcutaneously.

The postmortem pictures of the animals that died following deposition of segments of intestine and pieces of spleen of liver in their peritoneal cavities were much the same. Three or four hundred cubic centimeters of a brownish or serosanguineous fluid was usually present, and the peritoneal surfaces were reddened. The tissue placed in the peritoneal cavity could frequently not be identified other than as a friable, mushy mass. When the animal died the day after the operative procedure, or on the following day, the tissue was often spongy and contained gas. A fibrinous-like exudate was found uniformly about the tissue introduced. Cultures were made in several instances. In a number of these cultures *B. coli* were grown,

but with no greater regularity from the peritoneal cavity of the dogs in which intestinal segments had been placed than when liver or spleen was used.

In these experiments on the autolysis of various tissues in the peritoneal cavity, the liver and the intestine seemed to be the most toxic. Kidney tissue appeared relatively nontoxic. The disintegrating splenic tissue seemed intermediate in toxicity. The outcome in the dog in which the spleen was allowed to autolyze appeared to be affected favorably by the subcutaneous administration of salt solution. In the instances in which the liver and the intestine were placed in the peritoneal cavity, saline did not appear to have any value. It must be pointed out, however, that dogs with segments of intestine or liver in their peritoneal cavities lived only about a day, whereas the animals in which the spleens had been detached from the source of blood supply and which did not receive saline survived for more than three days.

When the autolysis was carried on in vitro in the incubator at 37 C. and the autolyzed tissue injected into the peritoneal cavities of rats at varying intervals, results of the same nature were obtained. The kidney and splenic tissues were definitely the least toxic. Bowel tissue, pancreas and liver appeared to be most toxic. The latter tissues seemed to disintegrate into toxic bodies more quickly than kidney and splenic tissue. After four days of incubation, the intraperitoneal injection of splenic tissue into a rat elicited marked symptoms. After four months' incubation, marked symptoms were elicited in all instances on injection. The injected substances appeared about equally toxic. Therefore, there would appear to be a qualitative difference in the toxicity of disintegrating tissues early in the process as well as a difference in the rapidity of autolysis.

Chemical studies of the urine of dogs in the peritoneal cavities of which tissues deprived of their blood supply were allowed to disintegrate showed a markedly increased excretion of nitrogen as was observed in the animals with simple occlusion of the intestine. The marked increase of blood urea nitrogen noted in animals with high intestinal obstruction was not observed in these; only slight increases in the nonprotein blood nitrogen were usually obtained.

Studies of the blood and urine nitrogen were also made in four dogs with irrigated obstructions of the closed loop in which the continuity of the intestinal tract had been reestablished by lateral anastomosis. These animals died from gangrene and perforation of the closed loop.[5] In three

5. Sixteen of twenty-five dogs with irrigated closed loops on whom Dragstedt and his co-workers experimented lived for some time. Possibly, a longer loop would diminish the danger of gangrene and perforation. At one time it was thought that this was a safe way to dispose of segments of intestine in the

TABLE 2.—*Toxicity of Various Tissues in Process of Autolysis*

Dog	Nature of Procedure	Day After Operation	Blood Mg. per 100 Cc.		Urine and Vomitus*	
			Urea	Chlorides	Amount	Nonprotein Nitrogen
55	Spleen detached from blood supply 10/4/26; no saline	0	16.80	480	.....	....
		1	14	330	.....	....
		2	14.54	410	.....	....
		3	10.26	430	.....	....
		4	20.53	400	.....	....
		5	28.93	360	.....	....
Dog died 10/9/26						
59	Spleen detached from blood supply 10/12/26; 400 cc. 2% sodium chloride given once daily	0	15.86	430	.....	....
		1	15.40	450	.....	....
		2	12.60	430	.....	....
		3	13.06	530	.....	....
		4	17.73	460	.....	....
		5	11.20	430	.....	....
	Saline discontinued	6	14	400	.....	....
		7	20.53	510	.....	....
		8	15.86	530	.....	....
		9	13.06	510	.....	....
		10	14.93	360	.....	....
		11	14.47	310	.....	....
Dog well						
113 Weight, 25 pounds (11.3 Kg.)	52 Gm. of intestine put into the peritoneal cavity 2/1/27; no saline	0	13.06	490	.....	....
		1	16.80	380	550	4.67
		2	15.86	400	1,470	5.35
		3	16.30	460	1,990	7.06
		4	17.20	430	1,000	6.40
		5	15.80	380	1,070	4.58
		6	18.36	390	1,310	5.49
		7	16.59	360	1,230	5.66
		8	15.37	410	700	3.26
		9	17.62	380	450	2.48
Dog recovered						
121 Weight, 30 pounds (13.6 Kg.)	Two kidneys (60 Gm.) put into the peritoneal cavity 2/10/27; no saline	0	10.26	460	120	1.14
		1	[illegible]6	490	375	2.62
		2	[illegible]3	380	1,320	7.58
		3	11.20	430	1,550	5.81
		4	24.26	440	1,100	6.34
		5	18.30	410	900	5.76
		6	16.53	390	700	5.32
		7	14.78	420	520	4.63
		8	17.33	380	580	3.28
		9	15.24	430	340	2.30
Dog well						
117 Weight, 35 pounds (15.9 Kg.)	15 inch closed loop obstruction; continuity reestablished by lateral and anastomosis 2/9/27; no saline	0	9	420	100	1.25
		1	18.60	400	1,650	4.77
		2	15.86	490	2,850	5.94
		3	23.08	360	1,900	5.60
Dog died 2/12/27; loop markedly gangrenous; perforations on antimesenteric border; bloody fluid in peritoneal cavity and in the closed loop						
126 Weight, 35 pounds (15.9 Kg.)	15 inch closed loop obstruction; continuity reestablished by lateral anastomosis 2/14/27; 400 cc. 2% sodium chloride once daily, discontinued on 2/17/27	0	14.70	430	110	1.02
		1	12.60	410	2,800	4.40
		2	16.80	420	4,700	5.95
		3	10.33	390	1,900	5.36
		4	14.47	640	475	4.41
		5	15.40	490	500	4.64
		6	13.52	450	340	3.27
		7	20.53	440	370	3.06
		8	24.26	410	260	2.87
Dog moribund; killed 2/23/27; perforations and extensive gangrene of closed loop						

* The dogs were allowed to drink water. They were starved for one day before being placed in metabolism cage and were not given food while in the cage.

"short circuiting" method for treating subjects with chronic intestinal obstruction. Experimental as well as clinical evidence has shown it to be decidedly unsafe. Perforations have been known to occur years after turning in the ends of such a loop. (Barracz, R.: Experimenteller Beitrag zur Frage der Totalen Ausschaltung mit totalem verschlusz der ausgeschalteten Darmschlinger zugleeich Beitrag zur axialen Darmvereinigung, Arch. f. klin. Chir. **58**: 120, 1899. Kammerer, F.: Bilateral Exclusion and Occlusion of the Intestine, Ann. Surg. **61**:497, 1915. Wiesinger: Zur Darmausschaltung, Deutsche Ztschr. f. Chir. **100**:1267, 1909.)

dogs, isolated loops were similarly irrigated, although the ends were left open and lateral anastomoses made; these dogs recovered. A second operation was performed later on one of these dogs; the ends were found partially sealed. There was free fluid in the peritoneal cavity. The dog died following a free opening of this loop. When extensive gangrenous changes obtain in irrigated closed loops, strangulation of intestine has occurred minus the obstructive factor. In these animals, an increased urinary excretion of nitrogen was obtained, but the high blood urea observed in simple occlusion of the upper part of the intestine was not noted.

Corroboration of the rapid disintegration of strangulated intestine previously noted [1] when a segment was strangulated with its mesentery and the constriction subsequently released was observed when the intestine was allowed to autolyze in vivo in the peritoneal cavity, or when the products of autolysis were injected into the peritoneal cavity of rats after being incubated in vitro. Comparisons made with other tissues showed the products of autolysis of intestine to be markedly toxic. Mann,[6] in removing the liver in dogs, has observed that when a piece of liver two fingerbreadths in width remained free in the abdomen, the dogs died within sixteen hours. Mason and Davidson [7] also observed the marked toxicity of liver tissue allowed to autolyze in the peritoneal cavity. None of the fifteen dogs survived in which from 30 to 110 Gm. of liver tissue was placed in the peritoneal cavity. The usual period of survival following the operative procedure was about twenty hours. Only three [8] of six dogs died in which the spleen used weighed from 23 to 68 Gm. The postmortem appearance of the peritoneal surfaces following the autolysis of tissue described in this paper was noted by these authors.[7] They also observed that the fluid present in the peritoneal cavity at death was much less toxic on injection than the autolyzing tissue itself.[9] Delbet and Karajonopoulos [10] made the same observation on muscle tissue autolyzed in vitro.

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6. Mann, F. C., quoted by Mason and Davidson: A Study of Tissue Autolysis in Vivo; I. Blood Changes, Physical and Chemical, J. Lab. & Clin. Med. **10**:622 (May) 1925.

7. Mason, E. C., and Davidson, E. C.: J. Lab. & Clin. Med. **10**:622 (May) 1925.

8. Mason, E. C., and Davidson, E. C.: A Study of Tissue Autolysis in Vivo; III. Observations Using the Spleen, J. Lab. & Clin. Med. **10**:997, 1925.

9. Mason, E. C., and Davidson, E. C.: A Study of Tissue Autolysis in Vivo; 11. A Pharmacological Study of the Toxic Material, J. Lab. & Clin. Med. **10**: 906, 1925.

10. Delbet and Karajonopoulos: De la toxicite des autolysats musculaires au point de vue du choc, Bull. Acad. de méd. **80**:13 (July) 1918.

Duval and Grigault,[11] studying the disintegration of protein in traumatic shock, observed a moderate increase in the nonprotein blood nitrogen of patients in a state of shock. Aub and Wu [12] obtained only slight increases in the nonprotein nitrogen of the blood in animals suffering from true shock after marked muscle trauma. Hashimoto [13] obtained moderate increases in the nonprotein nitrogen of the blood in which histamine intoxication followed repeated subcutaneous administration of histamine. Whipple and his co-workers [14] noted the increased urinary excretion of nitrogen following proteose intoxication.

In these experiments when disintegration of the tissue occurred following autolysis of tissue in vivo, a marked increase in the excretion of nitrogen in the urine was observed, with only a slight increase of the nonprotein nitrogen of the blood. The low values for blood chlorides noted in dogs with simple obstruction of the upper part of the intestine were not obtained in these animals. The increased urinary excretion of nitrogen is due probably only in a small part to the actual disintegration of the autolyzed tissue; the destruction of the devitalized tissue in all likelihood precipitates the breakdown of other body protein.

In this connection, it is interesting to speculate as to the cause of the marked increase in the blood nonprotein nitrogen observed in animals with simple occlusion of the upper part of the intestine. There is no evidence to indicate that the injury of the kidney is greater in animals with simple obstruction than in those in which tissue is allowed to autolyze. The rapidity with which tissue breaks down following dehydration and loss of chlorides would not alone account for the accumulation of nonprotein nitrogen in the blood, especially as this increase is dissipated by the administration of saline. The giving of saline does not prevent the increased urinary excretion of nitrogen, nor does the nonprotein nitrogen in the urine of these animals given saline seem to be increased over that observed in the animals that do not receive salt. Marshall and Davis [15] have shown that nearly all tissues have about the same urea content as blood. If an increase in the nonprotein nitrogen of the tissues in these animals occurred commensurate with that observed in the blood, if saline actually washed out the increased nonprotein

11. Duval, A., and Grigault, A.: L'intoxications par les plaies de guerre. La retention azotée des blessés, C. R. de soc. de biol. **81**:873, 1918.

12. Aub, J. C., and Wu, H.: Studies in Experimental Traumatic Shock: Chemical Changes in the Blood, Am. J. Physiol. **54**:416 (Dec.) 1920.

13. Hashimoto, H.: Blood Chemistry in Acute Histamin Intoxication, J. Pharmacol. & Exper. Therap. **25**:381 (June) 1925.

14. Cooke, J. V., and Whipple, G. H.: Proteose Intoxications and Injury of Body Protein, J. Exper. Med. **28**:223 and 243 (Aug.) 1918; ibid. **25**:461 (March) 1917.

15. Marshall, E. K., and Davis, D. M.: J. Biol. Chem. **18**:53, 1914.

nitrogen of the blood, an appreciable increase in the urinary excretion of nitrogen would be observed over that obtained in animals that did not receive saline. Water alone fails to obviate this increase in nonprotein blood nitrogen. It would appear that an alteration in the permeability of tissue may occur in these animals such that the rapid dehydration and loss of chlorides incident to the obstruction may permit the accumulation of nonprotein nitrogen in the blood.

In the recovery of the animals following the detachment of the spleen and its autolysis in the peritoneal cavity after the subcutaneous administration of saline, it would appear that saline possesses a protective value in combating the toxemia accompanying the disintegration of tissue. It has been maintained that saline performs such a function in the treatment of burns.[16] In the foregoing experiments, however, factors such as the protective influence of the omentum must be controlled before the recovery of the animal is ascribed to saline alone. Evidence was not obtained that would indicate that the administration of saline was of any value in combating the toxemia that occurred when the intestine was deprived of its blood supply.

SUMMARY AND CONCLUSIONS

Comparisons have been made of the toxicity of intestine and various tissues on autolysis in vivo and in vitro. The products of disintegration of the intestine were found to be markedly toxic when segments of small intestine were allowed to autolyze in the peritoneal cavity or when rats were given intraperitoneal injections of the products of autolysis of intestine from sterile containers kept in the incubator. A marked increase in the urinary excretion of nitrogen was observed in the animals in which various tissues were allowed to autolyze; however, only a slight increase in the nonprotein nitrogen of the blood occurred. The low values for blood chlorides observed in dogs with obstruction of the upper part of the intestine were not obtained in strangulation of the intestine alone (closed loop obstruction with gangrene) when the continuity of the remainder of the intestinal tract was restored. The subcutaneous administration of saline to animals with disintegrating segments of intestine appeared to be valueless.

16. Davidson, E. C.: Sodium Chloride Metabolism in Cutaneous Burns and Its Possible Significance for a Rational Therapy, Arch. Surg. **13**:262 (Aug.) 1926.

INTESTINAL TUBERCULOSIS CAUSING OBSTRUCTION *

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From April, 1927 to April, 1928 eight patients with hyperplastic intestinal tuberculosis with obstruction of varying degrees were operated on in the surgical service of the Peking Union Medical College Hospital. The cases, some of which have been reported in a short article previously published, form the basis of this report.[1]

Intestinal tuberculosis associated with pulmonary tuberculosis has long been recognized. The importance of early diagnosis and treatment in this condition has been recently stressed in a monograph by Brown and Sampson.[2] In 1925, Larimore and Fisher [3] reported a series of cases of ulcerative processes in the cecum in which the patients were operated on with encouraging though not brilliant results. In an article on tumors of the secum Erdmann and Clark,[4] in 1927, reported seven cases of tuberculosis of the cecum and terminal ileum with symptoms of intestinal obstruction. In this series, the pulmonary observations were negative.

FREQUENCY

Probably the most severe cases of pulmonary tuberculosis present some intestinal involvement; lesions are found in the intestinal tract of from 70 to 90 per cent of patients dying of this disease.[5] These lesions are usually of the ulcerative variety, and in only 25 per cent of them does stenosis or stricture develop;[6] it is during comparatively recent times that operation has been considered possible in such cases. The hyperplastic and stenosing lesions are more commonly treated surgically. In 100 cases of cecal lesions in which the patients were submitted to operation, Gant found four cases of hyperplastic tuberculosis; in Erdmann and Clark's series of forty-eight tumors of the cecum, seven were tuberculous.

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* From the Department of Surgery, Peking Union Medical College.

1. Branch, J. R. B.: The Surgical Treatment of Intestinal Tuberculosis, China M. J. **42**:239 (April) 1928.

2. Brown, L., and Sampson, H. L.: Intestinal Tuberculosis, Philadelphia, Lea & Febiger, 1926.

3. Larimore, J. W., and Fisher, A. O.: Tuberculosis of the Cecum, Ann. Surg. **83**:496 (April) 1926.

4. Erdmann, J. F., and Clark, A. E.: Tumors of the Cecum, Ann. Surg. **85**:722 (May) 1927.

5. Fenwick and Dodwell: Lancet **11**:133, 1892.

6. Read, W. D.: Tuberculosis of Cecum, Northwest Med. **20**:282 (Oct.) 1921; Cecal Pathology, ibid. **20**:11 (Jan.) 1921.

During the past five years, one of three resections of the right colon performed in this hospital was for tuberculosis. Within the past nine months, seventeen cases of intestinal tuberculosis have been seen on the medical service here, in which the condition was diagnosed by roentgen ray, but for one reason or another the patients were not transferred to the surgical service for operation. Although these figures are not intended to convey any information concerning the relative frequency of the condition in China, it is striking to see so many cases in Peking during one year. I did not encounter a case during twelve years spent in active surgical work in a clinic in Central China, though there must have been many in the community.

PATHOLOGY

About 85 per cent of the involvement in intestinal tuberculosis is in the terminal ileum, cecum and proximal colon.[7] Fifty per cent of the patients with tuberculous peritonitis have intestinal tuberculosis also, and 20 per cent of the patients having intestinal tuberculosis have peritoneal involvement as well.[8]

The type of lesion varies; in some instances, particularly when associated with pulmonary tuberculosis, it is ulcerative and progressive. This association with processes in the lungs and perhaps elsewhere is responsible for the term "secondary." Another type is the hyperplastic lesion, which is frequently called "primary." While it is true that in the latter variety there may not be any demonstrable tuberculous lesions in the lungs or elsewhere, and that from the data secured by physical examinations and roentgen-ray studies one may fail to find an original focus, it is, however, not only possible but highly probable that there may be or may have been processes elsewhere. Without complete examinations at autopsy, one is not justified in speaking of these cases as "primary."[9]

All of my cases were of the hyperplastic, stenosing variety, and the pathologic pictures were all strikingly similar (fig. 12). The lesions were in the terminal ileum, cecum and ascending colon; in several cases the appendix also was involved in the tuberculous process. Two were associated with tuberculous peritonitis, one in the ascitic and one in the early plastic stage. Three patients had evidence of previous pulmonary tuberculosis, but in only one was the process active. One of the speci-

7. Powell and Hartley: Diseases of the Lung and Pleura, Philadelphia, P. Blakiston's Son and Company, 1921, p. 798. Blumberg, Alfred: Pathology of Intestinal Tuberculosis, J. Lab. & Clin. Med. **13**:405 (Feb.) 1928.

8. Albrecht: Ueber die Beziehungen zwischen peritoneal und genital Tuberculose, Verhandl d. deutsch.

9. Charbrut, R.: Sur les risultats de la resection dans la tuberculose ileocaecale (40 observations), J. de chir. **30**:656 (Dec.) 1927.

mens showed a coexisting old infection with *Schistosoma* in the cecum and ileum, associated with tuberculosis.

The outstanding feature in all of the cases was a generalized thickening of the entire wall of the bowel with old, healed, scar tissue resulting in stenosis of the lumen; in several cases, the stricture was 5 mm. or

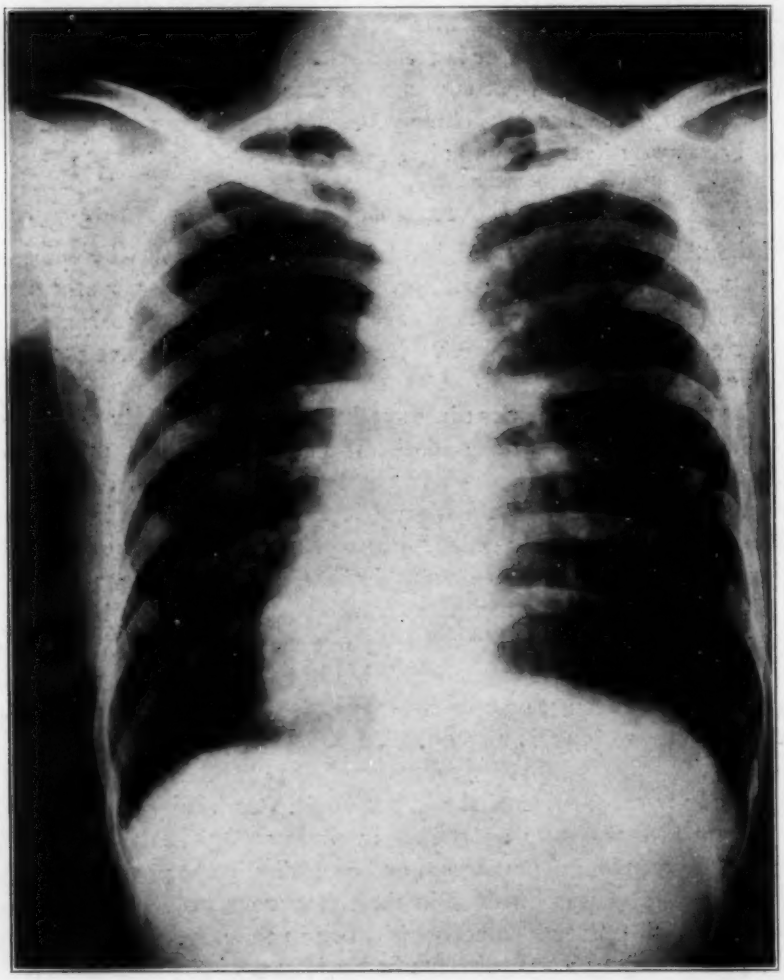

Fig. 1 (case 2).—Roentgenogram of the chest, July 19, 1927, showing thickening of apical pleura on the left side and fibrosed tuberculous infiltration of upper area on the right side. Lesions have not changed in six years.

less in diameter. The mucosa was thickened and often projected into the lumen as papillary elevations, some of them being from 1 to 2 cm. in length. Ulceration was not prominent, though in most of the cases small healed and unhealed ulcers were found. Grossly, little was seen

to suggest more than a determined and successful reaction to combat a chronic infectious process, so that the pathologist was disinclined to agree with the clinical diagnosis of tuberculosis until sections were examined. The gross pathologic picture would have been the same if the causative agent had been dysentery, *Schistosoma* or any other chronic

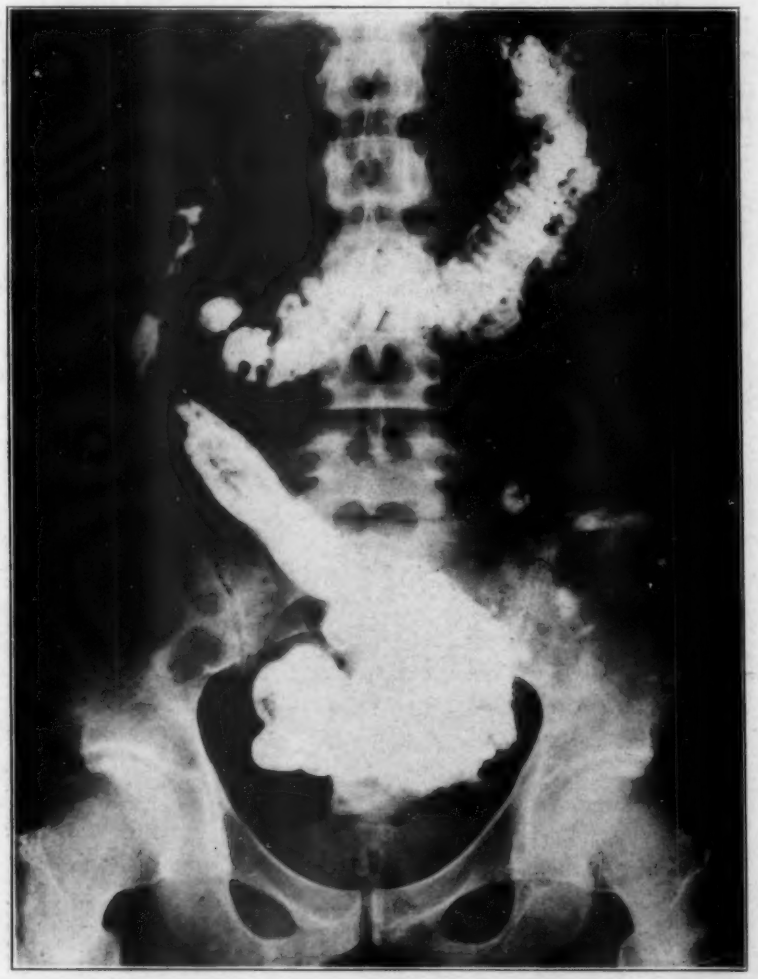

Fig. 2 (case 2).—Roentgenogram, July 19, 1927, made six hours after ingestion of barium, showing dilatation of the terminal ileum, nonfilling of the cecum and incomplete filling of the ascending colon.

irritant. The condition in all cases with the exception of one was diagnosed as tuberculous on microscopic examination, tubercles and bacilli being demonstrated. In case 6, essentially like the others clinically, and with similar gross pathologic changes, tubercle bacilli were not found

in the numerous sections examined. When evidence of dysentery or other probable cause for the lesion is lacking, I am of the opinion, however, that the patient also had intestinal tuberculosis, and the case is accordingly included in this series.

In some instances glandular involvement was observed; it was extensive and marked in one case. The complete report of case 5 is given

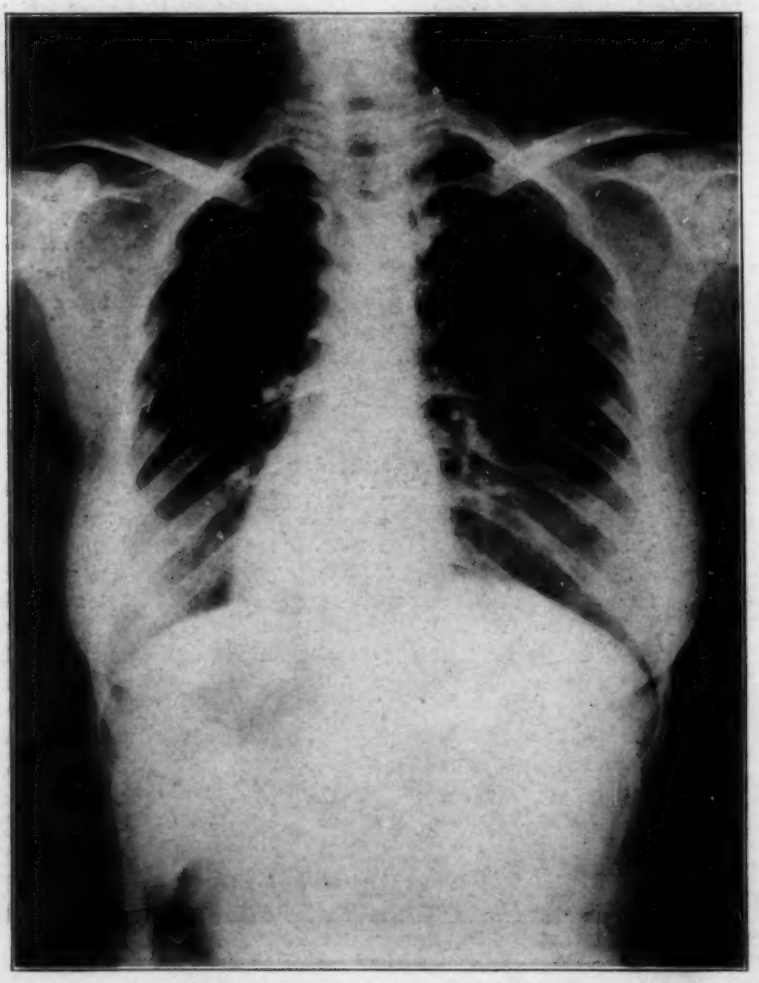

Fig. 3 (case 3).—Roentgenogram of the chest, Sept. 1, 1927, showing essentially normal lungs.

subsequently as typical of the observations in this series; the specimen removed at operation (ileum, cecum and ascending colon) is illustrated in figure 12.

REPORT OF CASE

At operation on a Chinese woman, aged 28, a specimen consisting of a portion of the small and large intestines, measuring 19 cm. in length and from 2 to 3 cm. in diameter, was obtained. Gross examination revealed a large amount of grayish, pink and light yellow fat and fibrous tissue attached to the external surface. The cut surface of the large intestine revealed an obstructive portion in which the lumen measured approximately 0.5 cm. in diameter. The mucosa in this region

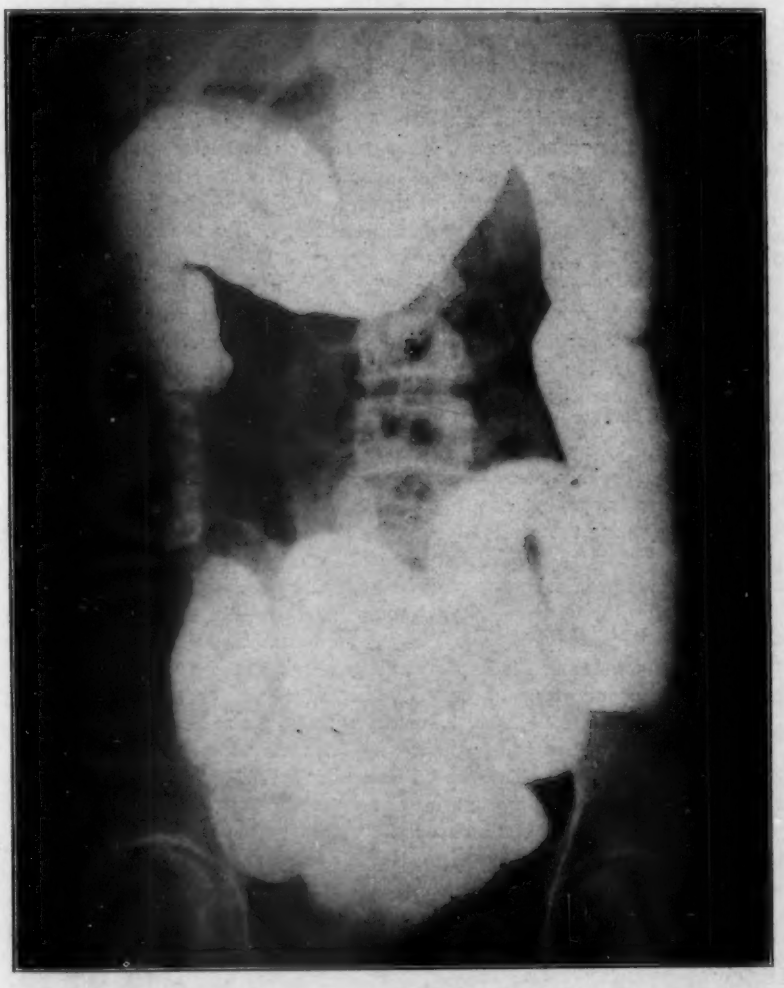

Fig. 4 (case 3).—Roentgenogram of the colon, June 28, 1927, after injection of barium enema, showing nonfilling of the cecum, general narrowing of the ascending colon at its proximal portion, incompetence of the ileocecal valve and dilatation of the terminal ileum. Diagnosis of a hyperplastic and obstructive lesion (tuberculosis) of the ileocecal region and the ascending colon was made on this examination.

showed many small and large papillary growths, varying in length from 0.1 to 1 cm. and in diameter from 0.1 to 0.3 cm. Near the papillary region, namely, in the large intestine, there were several grayish pink and dark red ulcers from 0.2 to 1 cm. in diameter. The mucosa in this region was swollen and elevated, and was a grayish white. The walls of the intestine varied from 0.3 to 2 cm. in thickness.

Sections of the small intestine examined microscopically revealed the irregularly thickened and edematous mucosa with infiltration by polymorphonuclear leukocytes, wandering cells, plasma cells and lymphocytes. Approximately in the center, there was a necrotic and hemorrhagic area which extended even into the submucosa. Elsewhere in the submucosa, marked edema and hemorrhage with infiltration by leukocytes was revealed. The muscularis was thickened in places and at one end showed edema with infiltration by a small number of leukocytes. Opposite the necrotic and hemorrhagic area, the muscularis was replaced by fibrous tissue with small and large blood vessels and infiltration of leukocytes. The serosa was thickened with a large amount of fat tissue and slight infiltration by leukocytes. In the mucosa, submucosa, muscularis and serosa, a few tubercles consisting of epithelioid cells and giant cells were present.

Sections of the large intestine showed that the mucosa was irregularly thickened and edematous with infiltration by wandering cells, plasma cells, lymphocytes and polymorphonuclear leukocytes. In places the submucosa revealed marked edema with infiltration by leukocytes. The muscularis was also irregularly thickened, and at one side areas of small and large intestinal glands were present, some of which were dilated and showed cystic structure, with lumina which were empty or contained a small amount of coagulated material, desquamated epithelium and many polymorphonuclear leukocytes. The muscularis was infiltrated with wandering cells, lymphocytes, plasma cells and polymorphonuclear leukocytes, especially surrounding those areas. Nearly a large tubercle consisting of epithelioid cells and giant cells was present. The serosa was also thickened and edematous with a large amount of fat tissue and infiltration by leukocytes. Sections of surrounding fat and fibrous tissue showed that many small and large tubercles consisting of epithelioid cells and giant cells were present. The tubercles were necrotic in a few places, containing dead cells and polymorphonuclear leukocytes, and were surrounded by lymphocytes and plasma cells. Elsewhere the fat and fibrous tissue also showed infiltration by lymphocytes, plasma cells, polymorphonuclear leukocytes and large cells with granular cytoplasm and oval or round nuclei in the center or near the edge. Here and there, slight hemorrhagic areas were also present. On careful examination only four tubercle bacilli were found. A diagnosis was made of tuberculosis of the cecum with stenosis of the lumen of the intestine.

CLINICAL PICTURE

My experience is similar to that of Erdmann, who stated that the condition in all of his seven cases had been diagnosed at various times as appendicitis. The patient in case 1 was operated on through a McBurney incision for appendicitis when the tuberculous cecum was found; in case 3 the patient had undergone appendectomy four months previously (the appendix was tuberculous); in case 5, exploration had been performed owing to severe, paroxysmal, abdominal pain thought to be due to a strangulated ovarian cyst; in the others, the condition at one time or another was thought to be appendicitis.

The predominating symptom is abdominal pain, at times general, at other times localized in the right lower quadrant. It is described as colicky, recurrent and paroxysmal, and, in some instances, is exceedingly severe; gurgling of gas is frequently present in the bowels, and in every case there is nausea and vomiting. The attacks of pain in some cases lasted three or four hours, in others as long as seven or eight hours. Most of the patients gave a history of constipation, and some of them had had attacks of diarrhea. In the interim they either were well, or

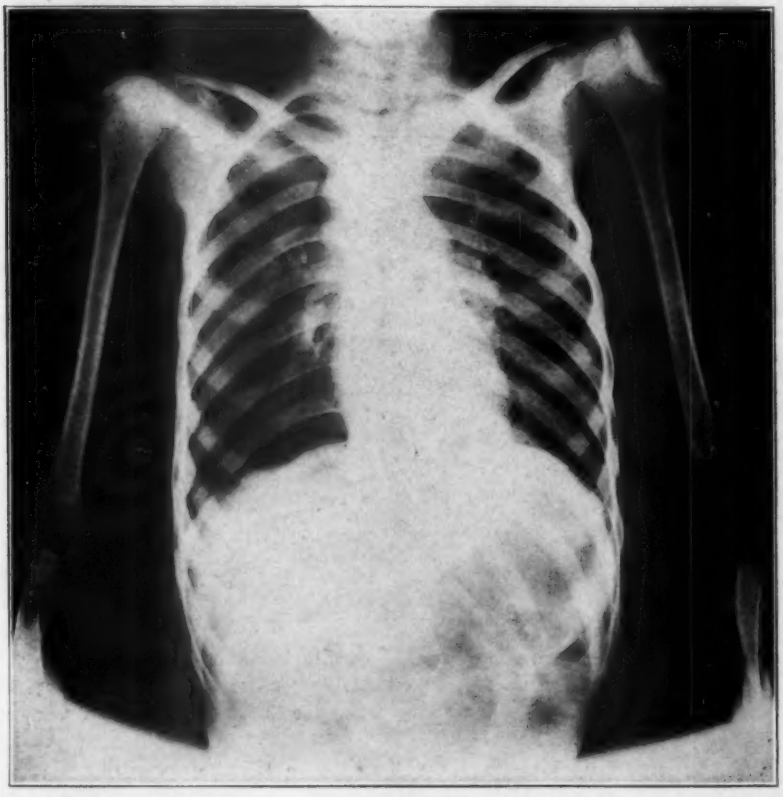

Fig. 5 (case 4).—Roentgenogram of the chest, Aug. 29, 1927, showing moderately increased thickening of the shadows of the hilum on both sides, which, in a subject aged 11 years, was thought to be of some clinical significance.

had vague abdominal discomfort; however, there was a definite tendency for the interval of freedom from attacks to become shorter and for the duration of the attacks to become longer. Such a symptom-complex would seem to indicate a pathologic process in the right lower abdominal quadrant, and one would naturally think first of the most common condition: appendicitis.

The physical, laboratory and roentgen-ray examinations are more illuminating. In my patients the chest either was normal or, as in three cases, showed old, healed, unimportant pulmonary lesions. In case 7 active lesions were seen. (These observations were confirmed by roentgen-ray films.) The abdominal examination did not reveal anything of importance except in the right iliac fossa; here localized, moderate tenderness was noted, but muscle spasm, involuntary resistance or rigidity was not present. In every case there was a mass in the cecal region which persisted after satisfactory enemas had been given. This mass had the limits of motility one might expect in the cecum, though in case 5 it could be pushed up to the costal margin and down to the pelvis. In one case with associated tuberculous peritonitis, the temperature was

Fig. 6 (case 4).—Roentgenograms, Aug. 31, 1927, made under fluoroscopic control following injection of barium enema, demonstrating the different phases of filling of the cecum and the ascending colon due to active spasm of these parts.

39.2 C.; in another, it was 38.8 C., and in a third uncomplicated case, it was 37.4 C. In the other cases the temperature was normal. The leukocyte count was normal in all but two patients; the patient in case 6 had 11,000 white cells with 86 per cent polyphonuclears and the patient in case 7 had 14,400 white cells with 74 per cent polymorphonuclears.

The roentgen-ray studies were most important. In all of the cases, with the exception of the first, roentgen-ray examination was made of the intestinal tract. The fluoroscopic and roentgenographic observations were characteristic of either ulcerative or hyperplastic lesion and in every instance, with the exception of case 8, confirmed the clinical diagnosis of intestinal tuberculosis. The constant symptoms were: (1) filling defect—nonfilling, irregularity, narrowing and constriction; (2) gen-

eral colonic hypermotility, and (3) ileal stasis. It may be worth while to mention here that all these observations are but the signs of ulceration or infiltration of the wall of the intestine, and not one of them offers explanation concerning the cause, which should be determined in the light of clinical and laboratory evidence.

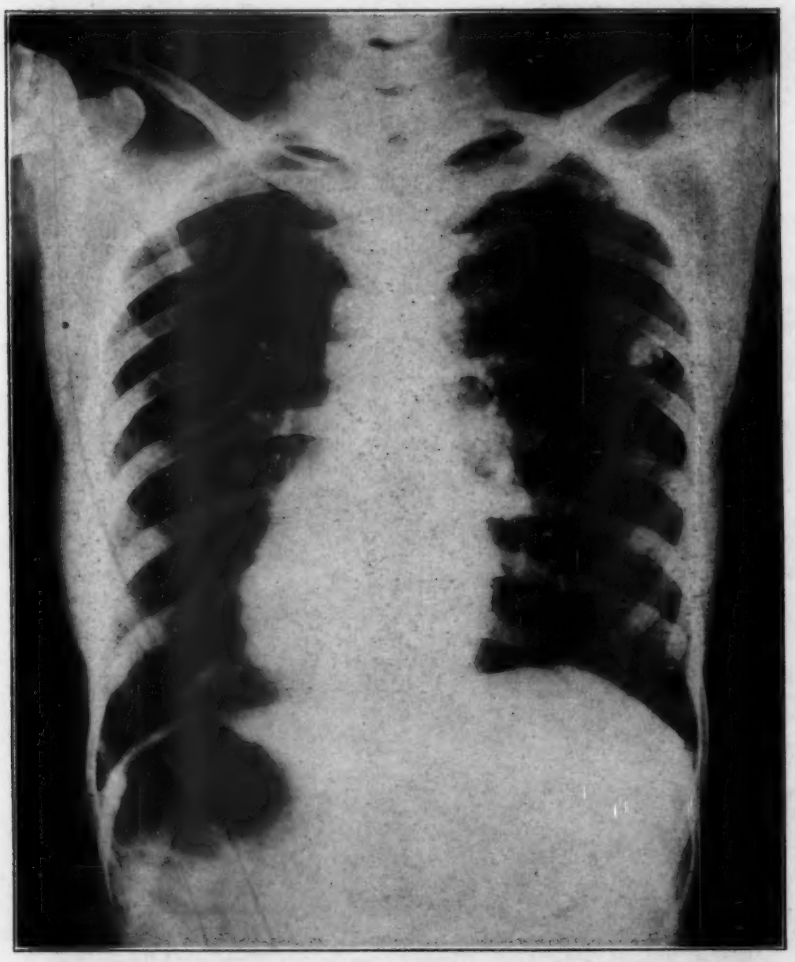

Fig. 7 (case 5).—Roentgenogram of the chest, Nov. 9, 1927, showing fibrosed lesion of lung in first intercostal space area and a calcified mass beneath the third rib near the axilla on the right side. Observations are considered as unimportant.

The greatest amount of information was obtained from examination following both a barium meal and an enema. In one case, the barium enema alone demonstrated the filling defect of the cecum and ascending colon clearly enough for a positive diagnosis to be made. Of the two

methods barium meal proved preferable because it provided the means of studying both the anatomic and the physiologic changes in the intestine, and, therefore, all the diagnostic signs, when present, could be seen. In all five cases in which the barium meal was employed, colonic hypermotility and ileal stasis, the cardinal sign-complex of ulcera-

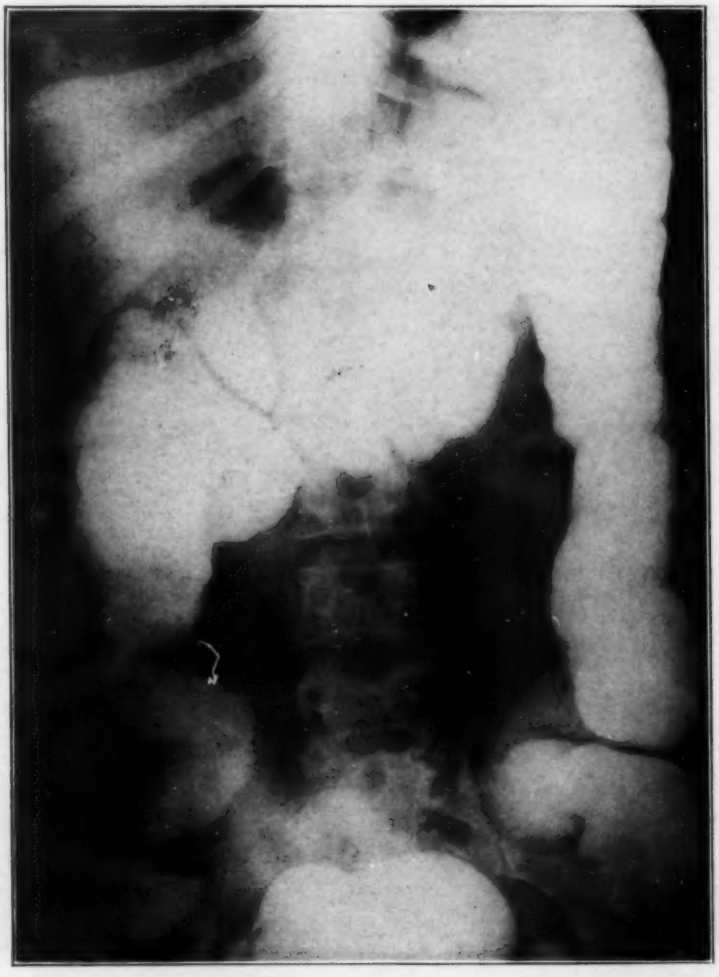

Fig. 8 (case 5).—Roentgenogram, Nov. 11, 1927, made after the injection of a barium enema, showing filling defect of the cecum and ascending colon, incompetency of the ileocecal valve and dilatation of the terminal ileum.

tion, were present in addition to the filling defect of the cecum and, in some cases, the terminal ileum. Localized tenderness was present in three patients, and a palpable mass which corresponded to the poorly filled cecum was observed in all the patients.

DIAGNOSIS

The two conditions with which one is most likely to confuse these hyperplastic occlusive lesions of the cecum are: appendicitis and malignant growth.

A malignant growth is usually seen in older people; it is most often associated with blood and mucus in the stools, and the symptoms are generally of short duration. My cases were all in young people between 25 and 30 years of age, and a malignant tumor was not probable. During the time in which this series of cases was collected a patient with a malignant growth in the right colon was not encountered.

The difficulty, therefore, has been to exclude appendicitis, and since the first two cases I have been moderately accurate in diagnosis. As already stated, all of my patients were admitted with a provisional diagnosis of appendicitis. If the history and clinical picture aroused suspicion of intestinal tuberculosis, a series of roentgen-ray pictures was made. One must, however, constantly have in mind the danger of this delay if one is dealing with a case of acute appendicitis. Three of my patients subjected to roentgen-ray examination were found to have chronic appendicitis with normal cecums.

If a patient with a suggestive history has a mass in the cecal area, a normal or nearly normal temperature, pulse rate and white blood count, no acute tenderness and no involuntary muscular resistance or rigidity, I have felt safe in carrying on the roentgen-ray studies of the intestine. Hourly counts were made in one case with leukocytosis, and the temperature was closely watched.

In case 8, the clinical diagnosis of intestinal tuberculosis was not confirmed by roentgen-ray studies but was demonstrated at operation; this case was in an early stage; with most of the lesion in the appendix and head of the cecum only. In another case of subsiding appendicitis, the roentgen-ray report indicated a hyperplastic occlusive lesion in the cecum; however, a lesion was not found at operation. With these two exceptions the roentgen-ray studies have not failed to give an accurate idea of the condition.

TREATMENT

Resection of the diseased portion of the bowel with enteroclostomy is the operation of choice; this was done in six cases, with one death and five recoveries resultant. I have developed an aseptic technic based on the method of Scarff,[10] and adapted it to end to side anastomosis; this has been described and illustrated in detail elsewhere.[11] Sometimes the

10. Scarff, J. E.: Aseptic End and End Suture of Intestine, Ann. Surg. **83:** 490 (April) 1926.

11. Branch, J. R. B.: Aseptic Intestinal Anastomosis in Resections of the Large Bowel, Surg. Gynec. Obst., 1928.

Data of Patients with Intestinal Tuberculosis with Obstruction

Case	Age	Sex	Duration of Symptoms	Previous History of Tuberculosis	Nausea and Vomiting	Pain	Leukocytes	Fever	Lungs	Abdominal Examination	Peritoneum	Operation	Complications	Pathologic Observations	Results
1	25	M	10 mo.	+	+	++	8,200	0	Latent tuberculosis in both upper lobes	Tenderness and mass in right lower quadrant; no rigidity	Normal	Resection of 30 cm. of terminal ileum and cecum; end to side enterocolostomy	Flare up of pulmonary process; superficial infection of incision	Tuberculosis of ileum and cecum, hyperplastic with ulceration	Cured
2	30	M	5 yr.	+	+	++	6,400-9,100; polymorphonuclears, 57-70%	0	Latent tuberculosis in both upper lobes; no advancement in 6 years	Tenderness and mass in right lower quadrant; no rigidity	Normal	Resection of terminal ileum, cecum and ascending colon; aseptic end to end enterocolostomy prophylactic ileostomy	Slight superficial infection around enterostomy tube	Tuberculosis and schistomiasis of terminal ileum, cecum and ascending colon	Cured
3	25	F	1½ yr.	None	+	+	4,600-5,300; polymorphonuclears, 68-73%	0	Clear	Tenderness and mass in right lower quadrant; no rigidity; small fecal fistula	Extensive tuberculosis of visceral and parietal peritoneum with ascites	Excision of fecal fistula; resection of 30 cm. of terminal ileum, cecum and ascending colon; enterocolostomy; prophylactic ileostomy	Phlebites of left femoral vein	Tuberculosis of ileum, cecum, appendix and ascending colon	Well enough to go to school but some vague abdominal discomfort
4	11	F	7 mo.	0	+	+	8,100; polymorphonuclears, 71%	39.2 C.	Negative on physical examination; X-ray shows thickening of shadows of the hilum thought to be of some importance	Doughy in consistency, and mass and tenderness in right lower quadrant; no rigidity	Extensive early plastic and ascitic tuberculosis of the visceral and parietal peritoneum	Exploratory laparotomy; enterostomy for obstruction	Flare up of pulmonary process; steady decline; intestinal obstruction	No autopsy	Died

5	28	F	4 yr.	0	+	++	6,800; polymorphonuclears, 74%	0	Clear	Tenderness and mass the size of an orange freely movable up to costal margin and down to pelvis; no rigidity	Normal	Resection of 15 cc. of terminal ileum, cecum and ascending colon; aseptic end to end enterocolostomy; prophylactic ileostomy	Slight superficial infection around enterostomy opening	Tuberculosis of the cecum and stenosis	Cured
6	28	F	8 yr.	0	+	++	11,000; polymorphonuclears, 86%	37.4 C.	Clear	Tenderness and mass in right lower quadrant; no rigidity	Normal	Resection of 15 cc. of terminal ileum, cecum and ascending colon; aseptic end to side enterocolostomy; prophylactic ileostomy	Slight infection of abdominal wall around enterostomy tube	Hyperplastic lesion of cecum and ascending colon, papillary hypertrophy of mucosa; no microscopic evidence of tuberculosis; guinea-pig inoculation negative	Cured
7	18	M	2 yr.	0	+	+	14,400; polymorphonuclears, 74%; 6,800	38.8 C.	Clear	Tenderness and mass in right lower quadrant; no rigidity	Normal; extensive and massive involvement of mesenteric and retroperitoneal glands	Exploratory laparotomy; the cecum and ascending colon are involved in a tuberculous hyperplastic lesion, and the last meter of the terminal ileum has scattered lesions	.....	Tuberculosis of mesenteric gland removed at operation	Unimproved
8	23	M	6 mo.	None	0	+	7,250; polymorphonuclears, 77%	37.2 C.	Pulmonary tuberculosis in both upper lobes	Tenderness and mass in right lower quadrant; no rigidity	Normal	Resection of 14 cm. of terminal ileum and cecum; aseptic end to end anastomosis; enterostomy (prophylactic ileostomy)	Pulmonary tuberculosis; bronchopneumonia; peritonitis from leak at site of anastomosis	Tuberculosis of cecum and ileum; chronic and acute appendicitis	Died

terminal ileum is as large as the colon, and end to end anastomosis is done. The patient in case 4 had also extensive tuberculous peritonitis and was not in condition to stand more than an enterostomy to relieve obstruction. The patient in case 7 was also in rather poor condition, and there was extensive and massive involvement of the mesenteric and

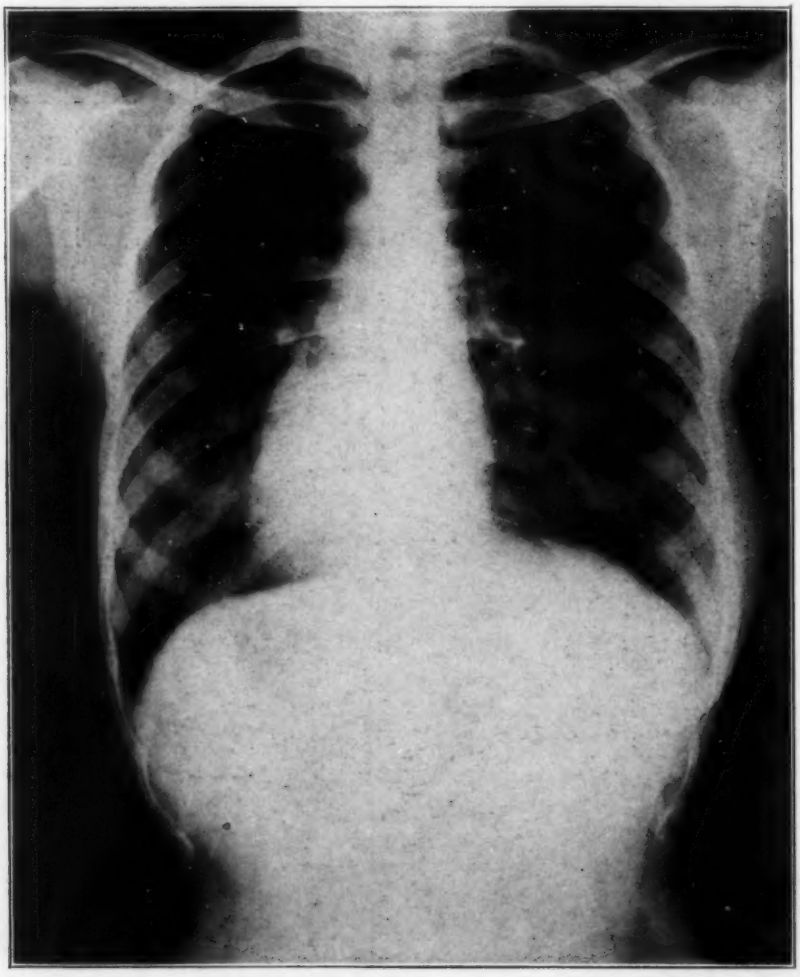

Fig. 9 (case 6).—Roentgenogram of the chest, June 3, 1927, showing normal lungs.

retroperitoneal glands, with caseation and softening; the abdomen, after exploration, was closed, without further treatment. In such a case, the surgeon might consider isolating the diseased portion of the bowel, leaving it in situ, bringing the two cut ends out through the skin, suturing them there and, at the same time, doing an enterocolostomy. When the

patient's condition permits it, this isolated section, by this time merely a mucous tube freed from the fecal current, may be excised. As the obstruction in my case of this type was not extensive, I decided to try general reconstructive treatment with heliotherapy and, later, resection. A simple short circuiting entero-enterostomy has not proved of great

Fig. 10 (case 6).—Roentgenogram, Dec. 6, 1927, made after the injection of a barium enema, showing no filling of the cecum and dilatation of the terminal ileum.

value. Considerable importance is attached to two features in the operative and postoperative procedure: (1) the prophylactic or complimentary enterostomy suggested by Charles Mayo to avoid possible paralytic

ileus and its consequences, and (2) the limiting, for four or five days after operation, of all fluid intake to the subcutaneous and intravenous administration of solutions of physiologic sodium chloride or dextrose. Nothing is given by mouth or rectum, and the liberal use of morphine further helps to reduce peristalsis to a minimum.

Fig. 11 (case 6).—Roentgenogram, Dec. 7, 1927, made eight hours after ingestion of barium, showing marked ileal stasis. The head of the barium column has reached the descending portion and sigmoid.

RESULTS

It is too early to report end-results in any of my cases. In the first five resections, there was no mortality; in the sixth, the patient died of pneumonia and peritonitis. The autopsy showed a leak at the anasto-

mosis with a resultant peritonitis. The area of cecal involvement was slightly greater than estimated at operation, and not enough of the bowel was resected; the sutures, not being taken in healthy normal colon, sloughed; the patient also had pulmonary tuberculosis and bronchopneumonia. In three resections, some local infection was present in the

Fig. 12 (case 5).—Specimen removed at operation, described in text.

abdominal wall around the enterostomy opening. This was probably due in part to two factors: (1) the use of catheters larger than necessary in the prophylactic enterostomy; (2) too abrupt removal which allowed the stab wound to close too soon with an infected path undrained. All my incisions healed by first intention, and in none was there evidence of peritoneal contamination, except in the fatal case. All patients passed

gas per rectum spontaneously, and stools were normal when food was given by mouth. The enterocolostomy openings functioned normally in all cases as shown clinically and by follow-up roentgen-ray examinations. One patient died—the patient in case 4, who had extensive intestinal tuberculosis of the early plastic stage in addition to the intestinal lesion. Emergency enterostomy was performed to relieve intestinal obstruction, and the patient died forty-two days after operation. Five patients are well enough to carry on their usual vocations.

MALIGNANT TUMORS OF THE WALL OF THE CHEST

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PASADENA, CALIF.

Malignant tumors of the wall of the chest, apart from metastatic carcinoma, are relatively rare. They are usually sarcomatous. Two hundred and thirteen cases were collected by Hedblom [1] in 1921; of these, 61.4 per cent were sarcoma and 18.7 per cent chondroma. Few cases appear in the literature since that time, and in view of the great interest which is now maintained in thoracic surgery, it seems worth while to report two cases in which the patients have been under my care in the past two years.

The pathologic processes of these tumors does not differ from that of sarcoma or chondrosarcoma in other parts of the body. They may take their origin from the soft parts, the cartilage, ribs, sternum, clavicle or scapula. As the growth develops, it invades all of the structures of the wall of the chest and may finally attack the pleura. The two cases here reported began in the cartilaginous portions of the upper ribs near the manubrium.

REPORT OF CASES

CASE 1.—Mrs. K., aged 50, childless, consulted me in November, 1922, concerning a tumor of the wall of the chest beneath the right breast. She first noticed the tumor in April, 1922. It was painless but seemed to be increasing in size. She had gained in weight. Examination revealed a bony-like tumor involving the third, fourth and fifth ribs on the right side near the costocartilaginous junction. It was slightly movable and not tender. The breast was normal; there were no enlarged axillary glands. Stereoscopic x-ray films showed an area of increased density, 8 by 6 cm. between the first and fourth ribs. There was no involvement of the ribs, and the tumor mass appeared to be anterior to them. No roentgenographic diagnosis was possible.

First Operation.—Nov. 28, 1922: The tumor was exposed by a transverse incision just below the clavicle. It proved to be cartilaginous, 6 cm. in diameter, lying between the first and second ribs, extending inward to the pleura and outward between the ribs for 2 cm. There was erosion of the first rib. The tumor was excised down to the ribs and then curetted out as far as seemed safe without penetrating the pleura. One hundred milligrams of radium screened with 2 mm. of brass was left in the cavity for twelve hours.

The patient remained well without recurrence until July, 1924, when recurrence took place near the sternal ends of the ribs. X-ray examination showed recurrence in the area of the original tumor. The patient did not suffer pain and there was no evidence of metastasis.

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1. Hedblom: Tumors of the Bony Wall of the Chest, Arch. Surg. **3**:56 (July) 1921.

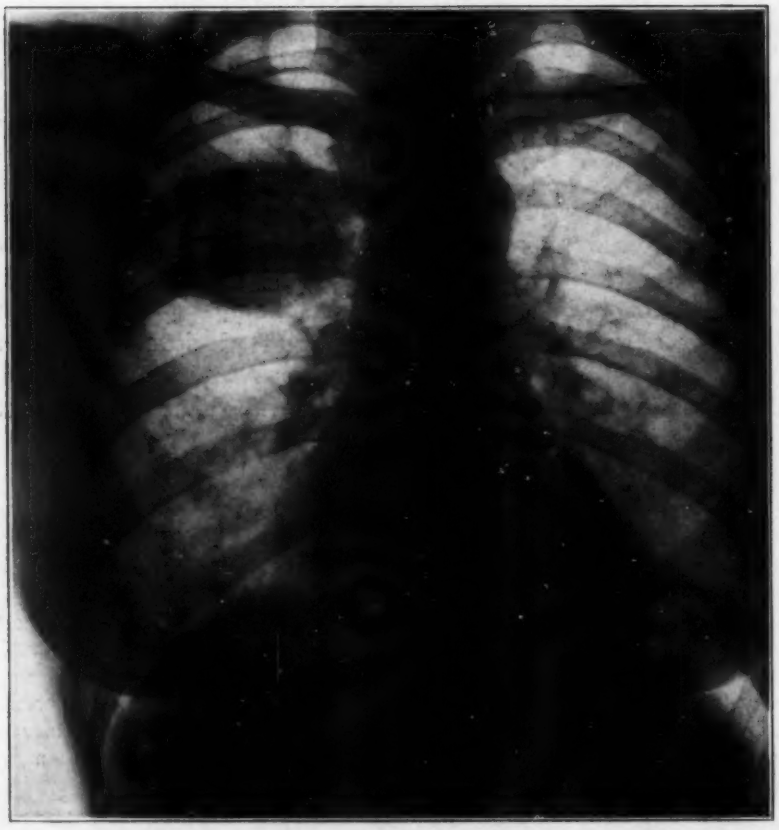

Fig. 1 (case 1).—Tumor of the wall of the chest; the area of density is shown extending from the second to the fourth ribs, near the sternal border of the right side.

Fig. 2 (case 1).—Recurrence of tumor at original site. The area is less dense.

Fig. 3 (case 1).—Lateral view of tumor.

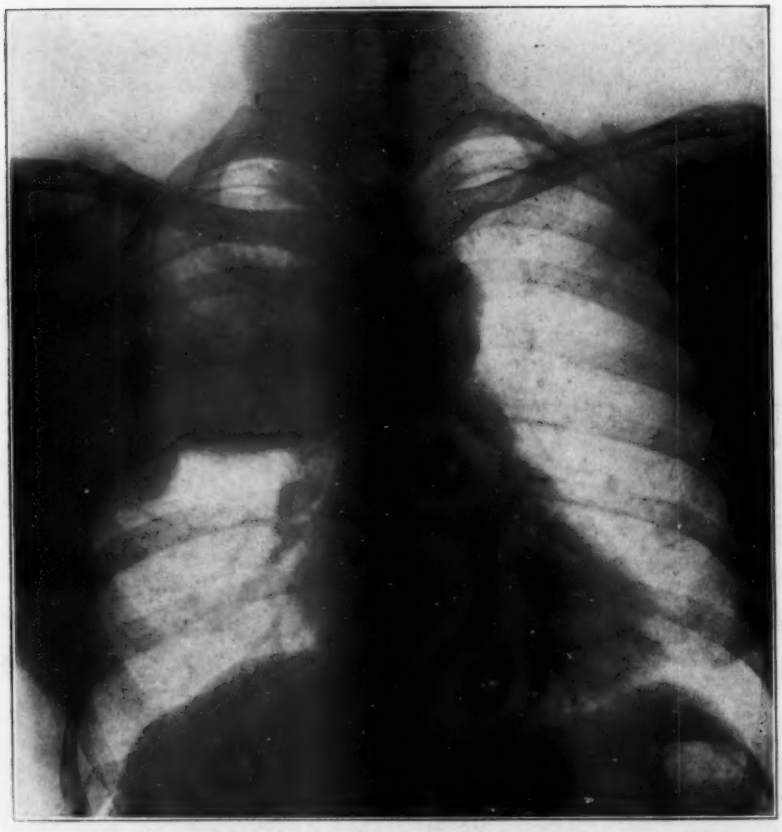

Fig. 4 (case 1).—Greatly increased area of density extending outward to lateral border of the chest.

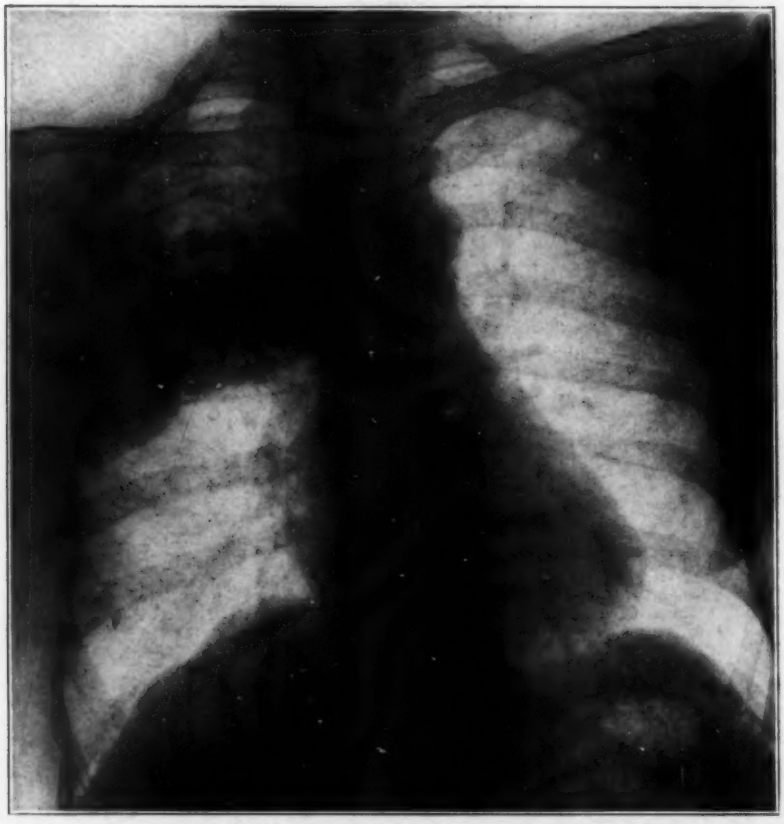

Fig. 5 (case 1).—View of tumor showing denser shadow but without apparent extension.

Fig. 6 (case 1).—Lateral view of tumor showing invasion of pleura and lung.

Second Operation.—July 21, 1924: The old scar was excised and the incision enlarged. The tumor had recurred at the original site. The first rib was eroded and destroyed for 2 or 3 cm. outward from its sternal junction. There were two or three recurrent nodules in the periosteum overlying the second rib. The second rib was removed for 2 inches (5 cm.), the first rib and sternum thoroughly curetted. The pleura was accidentally opened but no serious symptoms followed. Fifty milligrams of radium in bare needles were inserted into the area of the tumor. The radium was left in ten hours. The patient remained well until May 20, 1927, when she returned complaining of pain in the right arm and some restriction of motion. The tumor had recurred in the scar in the second rib farther out and beneath the clavicle. X-ray examination showed involvement of the second, third and fourth ribs.

Third Operation.—June 13, 1927: A large flap was made by a curved incision beneath the breast and pectoral muscles. The second, third and fourth ribs were

Fig. 7 (case 1).—Scar from operation; the right breast is slightly elevated.

divided at the sternocostal junction and the pleura opened to inspect the tumor. It had involved the third rib in the anterior axillary line and there was an extension within the chest cavity involving the second, third and fourth ribs and the parietal pleura. A tumor mass 5 or 6 inches (12.7 or 15.24 cm.) in diameter projected into the pleural cavity and was attached to the upper lobe of the lung. The tumor masses involving the ribs were all curetted and 50 mg. of radium in 12.5 mg. bare needles was inserted into the tumor mass within the chest cavity and left in twenty-four hours. The defect in the wall of the chest was closed by sliding up the breast and pectoral muscles. The wound healed without infection.

Present Condition.—Feb. 10, 1928: Clinically, this patient has remained well, but a recent examination revealed a tumor mass 2 cm. in diameter between the axillary folds, firmly attached to the wall of the chest. X-ray films do not show any increase in the size of the tumor within the chest cavity.

Pathologic Process.—Grossly, this tumor consisted of pearl-like cysts which formed pockets in the cartilage of the ribs and later invaded the bony portions of the ribs and manubrium.

Fig. 8 (case 1).—Tumor of the wall of the chest; chondrosarcoma; × 75

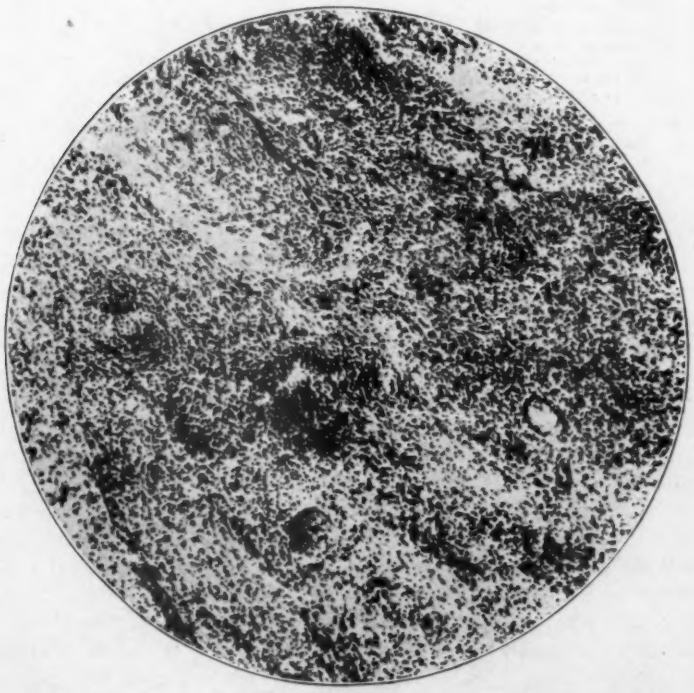

Fig. 9 (case 2).—Tumor of the wall of the chest; round cell sarcoma; × 75.

The microscopic report was made by Dr. A. M. Moody. The tissue consisted essentially of hyaline cartilage with many nuclei, having the appearance of dividing cells and some round cells. As a whole, it presented the picture of a malignant growth. A diagnosis of chondrosarcoma was made (figs. 1 to 8).

CASE 2.—A man, aged 26, a concrete worker, for one month prior to my first examination had noticed a swelling in the region of the right pectoral muscle just below the clavicle. Six years before, he was struck on the chest with a piece of iron, near the site of the present tumor. The scar of this injury is still visible. He was otherwise healthy. The results of the examination were negative except for the presence of a semifluctuating tumor, 6 cm. in diameter lying just beneath the right clavicle and extending from the outer border of the sternum to a line drawn from the central portion of the clavicle. The tumor was painless, some-

DATA IN CASE 2

	200 Kilovolts, 4 Milliamperes, 50 Cm. Distance, 18 Cm. Aperture, ½ Mm. Copper, 1 Mm. Aluminum Filter	
12/15/26	Upper anterior right side of chest	15 minutes
12/16/26	Upper anterior right side of chest	15 minutes
12/17/26	Upper anterior right side of chest	20 minutes
12/20/26	Upper anterior right side of chest	20 minutes
12/22/26	Upper anterior right side of chest	20 minutes
12/24/26	Upper anterior right side of chest	20 minutes
12/27/26	Upper anterior right side of chest	10 minutes
12/27/26	Right posterior angle of scapula	10 minutes
12/29/26	Right posterior angle of scapula	20 minutes
1/ 3/27	Right posterior angle of scapula	20 minutes
1/ 5/27	Right posterior angle of scapula	20 minutes
1/ 8/27	Right posterior angle of scapula	30 minutes
1/10/27	Right posterior angle of scapula	20 minutes
	Pain diminished; mass in back became smaller; open wound in anterior aspect of chest cleaner	
2/ 8/27	Right anterior side of chest	30 minutes
2/ 9/27	Right anterior side of chest	30 minutes
2/11/27	Right anterior side of chest	30 minutes
2/12/27	Right posterior side of chest	30 minutes
2/18/27	Right posterior side of chest	30 minutes
	Following first treatments pain ceased; general condition improved; wound in anterior chest wall closed in considerably; remaining area shows clean granulations.	

what movable and apparently attached to the underlying structures. X-ray films revealed an area of only slightly increased density at the site of the tumor.

Operation.—July 29, 1926: A curved incision over the wall of the chest exposed a tumor, 6 cm. in its long diameter, densely adherent to the third rib and involving its periosteum. The tumor was vascular and had infiltrated the pectoral muscles and passed between the third and fourth ribs, involving the parietal pleura. The tumor was excised as widely as possible; 50 mg. of radium screened with 2 mm. of brass was left in the wound for twelve hours. The wound did not heal readily. There was a free discharge of cheesy material and free hemorrhage. In September, 1926, 50 mg. of radium was placed in the cavity in bare needles for two hours. The wound improved much after this and granulations assumed a healthy appearance, but a pocket still persisted which continued to discharge and bleed.

Nov. 8, 1926: Four months after operation, 1,200 mg. hours of radium in bare needles were given in different portions of the tumor. There was a well marked constitutional reaction and the tumor shrank greatly. However, it continued to bleed freely and at each dressing fragments of grayish necrotic material escaped. At this time, a tumor appeared near the angle of the right scapula

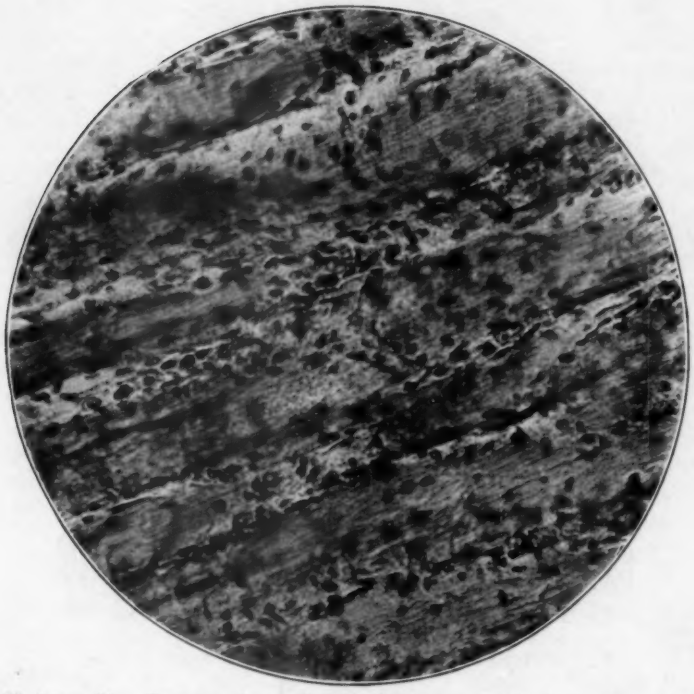

Fig. 10 (case 2).—Same as figure 9, showing more detailed structure; × 300.

Fig. 11 (case 2).—Infiltration of the muscle fibers; × 100.

Fig. 12 (case 2).—Sarcoma of the wall of the chest.

Fig. 13 (case 2).—Metastatic sarcoma of the back.

which was similar in appearance and consistency to the original tumor. The patient was now referred to the x-ray department for deep therapy. He was given treatments to the point of tolerance. He had a severe reaction from the x-ray therapy. There was marked improvement in the original tumor with diminished discharge and less hemorrhage. The tumor on the back almost disappeared.

Pathologic Process.—Grossly, this tumor consisted of grayish white tissue. It was soft, friable and seemed to infiltrate the muscle. There was no regular form or contour. In places, it seemed glandular (fig. 9 to 13).

Microscopically, it consisted of small round cells, infiltrating the adjacent muscle fibers. A diagnosis of round cell sarcoma was made.

Present Condition.—Feb. 10, 1928: The patient has lost 10 pounds (4.5 Kg.) in weight; he is anemic and weak. The tumor has not increased in size but it is still discharging, bleeds freely and has eroded the second rib. There is a dense fibrosis of the pectoral muscles, and he is unable to abduct his arm or lift it above his head. The tumor over the scapula has not recurred, but there is an ulcer 2 cm. in diameter at its former site. No metastasis can be discovered either by examination or on the x-ray films.

COMMENT

Tumors of the wall of the chest are usually malignant. The most common forms are sarcoma and chondrosarcoma. These tumors usually recur after removal. The patients are best treated by thorough removal, radium and x-ray therapy.

CRANIOTOMY INCISIONS WITHOUT FORCEPS*

ALBERT S. CRAWFORD, M.D.
DETROIT

The control of hemorrhage is one of the most important and yet difficult problems in all extensive craniotomies. The tourniquet, hemostatic stitch and pedicle clamp have been replaced largely by the multiple forceps method now commonly used. The covering of the scalp is generally accomplished by towels clamped or sewed to the cut edges.

During the past two years, my co-workers and I have been using a method which keeps the operative field free of forceps and which at the same time efficiently covers the scalp, controls bleeding and protects the edges of the incision. This is by the use of stockinet and Michel clips.

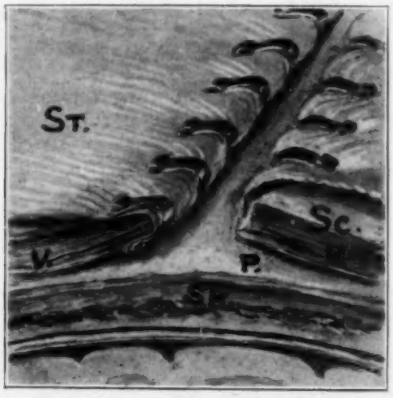

Fig. 1.—Placement of Michel clips on skin incisions. *ST*, indicates the stockinet covering; *Sc*, the scalp; *V*, the vein; *P*, the pericranial layer and *Sk*, the skull.

We experimented on various metal clips, but finally settled on the Michel type, for they are already available, are inexpensive, are easily applied and control hemorrhage efficiently.

A few points in the technic might be mentioned. After the field block has been made with procaine hydrochloride, the incision line is scratched till it bleeds, then the stockinet is laid over the whole operative field and cut along the blood-stained line. The skin incision is made in the usual manner, a few inches at a time, and bleeding is controlled by digital pressure. When a spurting vessel is encountered, it is caught at once and stick tied with silk. All other oozing is controlled with the clips. Figure 1 shows how the clips are placed, external to the

* From the Henry Ford Hospital.

pericranial layer, about 12 mm. apart and directly over bleeding points wherever possible. Figure 2 shows a temporoparietal incision all ready for the work on the skull, and figure 3 the clips, the holder and the forceps used.

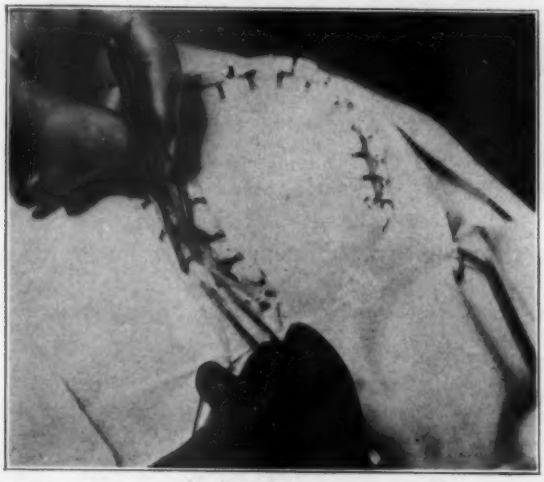

Fig. 2.—Temporoparietal incision with clips already placed.

Fig. 3.—Michel clips (small size) single (top and side views), on the wire holder, and one in the forceps which is used to apply (reduced one-half).

After the bone flap has been turned, the wound edges are covered with wet cottonoid sheets, so that the field is smooth and level and the clips are protected. During closure, the clips are removed a few at a time, as the skin sutures are placed and tied, to prevent excessive bleeding from the scalp.

Unfavorable results have not followed this method so far. Some sloughing of the skin edges occurred in one case, resulting in delayed union, but this was attributed to extremely low blood pressure following the operation and to probable thrombosis of the vessels. The clips, of course, may have been contributing factors. This danger from pressure should be kept in mind.

Essentially the same method was recently described for incisions on the extremities by Dr. W. E. Johnston.[1] We agree with him that the method can be effectively used for protection of wounds in all surgical incisions, except probably those leading to large cavities, such as in laparotomies, in which clips might be dislodged and lost. A refinement in technic might be elaborated by having the clips all fastened to a string or wire, which would eliminate the danger of any being lost.

1. Johnston, W. E.: Method of Operative Protection from Skin Infection, J. A. M. A. **88**:1320 (April 23) 1927.

FRACTURE DISLOCATION OF THE SHOULDER

RELATION OF SOFT PARTS TO RESTORATION: A NEW METHOD OF TREATMENT

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As a general rule, a fracture or dislocation is thought of by the general practitioner solely in terms of the relation that bones or fragments of bones bear to each other. The effect of the soft parts in restoring normal alinement or in preventing it is lost sight of.

The interposition of soft parts in preventing union, causing malunion or interfering with restitution is well recognized and will not be discussed. But the clinician with increasing experience is more and more impressed with the importance of the utilization of the closely encircling muscles, ligaments, tendons, capsule and periosteum or portions of the latter in marshaling fragments into alinement. Failure to consider this important factor often leads to poor end-results, disappointment to the attendant or serious disability to the patient.

In such complicated joints as those of the shoulders and hips, this is especially important, and a precise knowledge of the anatomy of the parts, the exact function of each muscle and the direction of the forces that produced the disability should be known.

In the treatment of patients with intracapsular fracture of the hip, Royal Whitman of New York places the patient in the now well recognized "Whitman position," and by extreme abduction makes use of the capsule put on the stretch to marshal the fragments into apposition. This is aided through fixation of the pelvis by abduction of the other leg and by traction and inward rotation of the affected one. In this connection, Lovett and Jones[1] say that "any treatment which ignores the fundamental principles of abduction, extension and internal rotation in intracapsular fracture of the hip, should be ruthlessly condemned as obsolete," but they fail to explain the modus operandi of the soft parts.

During the early stages of fractures and dislocations, muscle spasm is known to play an important rôle in preventing reposition, but its continuance after reduction is frequently overlooked. Thus, with the patient under an anesthetic, a fracture is reduced and the part involved placed in a carefully applied plaster cast, and the surgeon has a comfortable feeling that all is well. When the patient regains con-

1. Lovett and Jones: Orthopaedic Surgery, New York, William Wood & Company, 1923, p. 404.

sciousness and an x-ray picture is made, it is found, however, that the reestablished muscle spasm has again pulled the fragments out of alinement. The remedy is manifestly to be found in traction or extension to avail of not only marshaling the fragments by the encircling soft parts, but to overcome muscle spasm before final fixation. In such cases, the traction should be continued until the muscle spasm ceases. Fractures at the middle of the forearm, after reduction with immediate fixation, are most prone to get out of alinement from the continuance of muscle spasm of the pronators, with an ultimate lessening of the interosseous space and rotatory movements of the forearm, unless corrected early.

Economy in x-ray pictures, after reduction of fractures and prior to good callus formation, is false economy, and the relation of the fragments to each other should be frequently determined, not presupposed, especially in the early stages of healing.

In order to emphasize the points that I desire to bring out in this paper, it may be well to recapitulate certain points concerning aids in restitution which should be considered.

1. Traction on the surrounding encircling soft parts should be utilized to help assemble the fragments, in addition to any manipulation or leverage that the surgeon can effect from without.

2. The possibility of postanesthetic muscle spasm pulling the fragments out of alinement must be borne in mind. In suitable cases, it will be well to use traction until muscle spasm is permanently overcome.

3. Fixation should be used only after muscle spasm is overcome in cases in which muscle spasm can distort the parts.

4. X-ray pictures should frequently be made until callus formation assures the desired restoration. The surgeon should not be content with an x-ray picture before the cast or splint is applied and after it is taken off.

REPORT OF A CASE

In order to illustrate the foregoing principles, the following case will be cited as being typical.

H. C., aged 28, fell off a platform about 8 feet high (244 cm.) on June 27, 1926, striking on his left shoulder. A report of his condition at that time states that pain and crepitation were present. A depression was noted under the left acromion process, and there was undue fulness on the anterior aspect of the joint. The arm was rotated inward and abducted, and the forearm was supported by the right hand, near the elbow.

An x-ray picture made at that time showed a fracture dislocation at the proximal end of the left humerus. The dislocation was of the subcoracoid type, being anterior and inward from its proper position, so that the glenoid was not visible in the roentgenogram. The fracture was of the surgical neck, and from the

muscle spasm of the supraspinati and infraspinati the proximal fragment or humeral head had rotated through an arc of 180 degrees, so that its fractured surface was directed upward; the line of fracture of the distal end was horizontal and slightly below and external to the articular surface of the fractured humeral head (fig. 1).

Prior to the time the patient entered my service, an attempt had been made to reduce the fracture and dislocation while he was under an anesthestic, with the result that the distal fragment had slipped into the axilla and was abducted at an angle of 45 degrees with the body and was causing much axillary pain from impingement on nerves. The proximal end or head was unchanged in position. After this manipulation, it was held in position by a plaster cast covering the arm

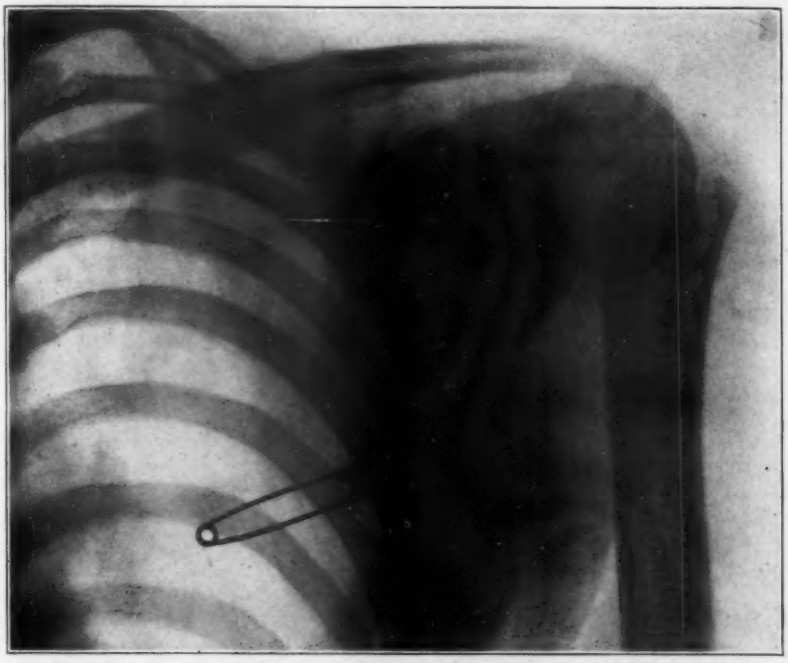

Fig. 1.—Fracture-dislocation of left shoulder immediately after accident.

and thorax (fig. 2). The patient was referred to me in this condition seven days after the accident, and was admitted to the hospital on July 2, 1926.

For this condition, various manipulative procedures of doubtful value have been suggested and tried, with the patient under general anesthesia; also similarly ineffective open-operative methods of grafting, suture, screwing the parts together, bone-pegging and even removal of the dislocated proximal fractured head. In these cases, it is wisest to put into practice the principles set forth in the first part of this paper. As in the present case neither the dislocation nor the fracture had been reduced by manipulation, the retaining cast was removed and the patient put up in a Balkan frame. He was placed flat on his back with the spring and mattress made additionally firm by cross-slats. Projecting from under the mattress beyond the left side of the bed for about 2 or 3 feet (61 or 91 cm.) was a board 5 feet (152 cm.) long and 4 inches (10 cm.) wide; to its upper surface at the

Fig. 2.—Fracture-dislocation in plaster cast after attempt at manipulative reduction.

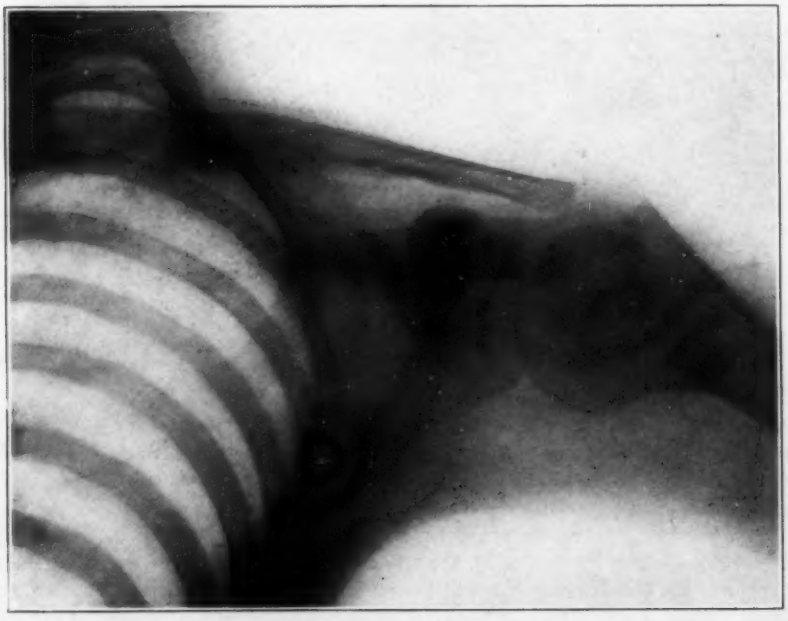

Fig. 3.—Forty-eight hours after traction (15 pounds) on distal fragment and suspension of 5 pounds each of the forearm and humerus to separate pulleys. Abduction of humerus 60 degrees. Dislocation reduced.

outer end a similar piece of board, about 4 by 4 inches was nailed at right angles; at its center it was surmounted by a vertical pulley, which was screwed in. By means of this rough board, which the mattress and the patient's weight held in any position in which it might be placed by the surgeon, horizontal traction over the pulley could be made at any desired angle of abduction by means

Fig. 4.—Position of patient in Balkan frame.

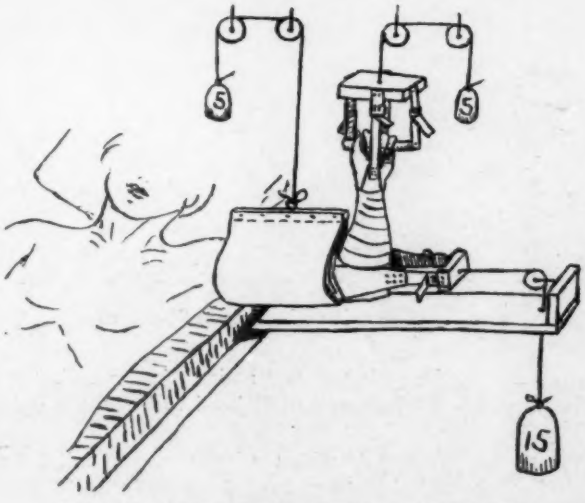

Fig. 5.—Diagrammatic drawing of apparatus.

of adhesive straps and a spreader attached to the upper arm. The forearm, by means of adhesive traction straps from elbow to hand, and the upperarm, supported by a sling with spreader, were suspended from the overhead bars of the Balkan frame, with appropriate pulleys and counter-balance weights. These enabled the patient to change his position within reasonable limits and facilitated nursing. About 5 pounds (2.3 Kg.) each were used as counterpoises for suspen-

sion of the forearm and of the arm, and in abduction at the side of the bed, from 10 to 15 pounds (4.5 to 6.8 Kg.) were used as the case progressed (figs. 3 and 4).

This apparatus was applied on July 2, and on July 6, the x-ray picture showed that reduction of the dislocation had been effected (fig. 5). Abduction of the arm at this time was about 45 degrees with the body, and it was seen that the apposition of the fragments was not in good alinement. It was therefore increased to 60 degrees and later to 90 and 120 degrees until perfect alinement was obtained (figs. 5, 6 and 7).

On July 28, after three weeks of treatment in bed, when the x-ray examinations showed good callus and a reduction of the dislocation and normal alinement at the fracture, the arm was carefully held in the position maintained by the

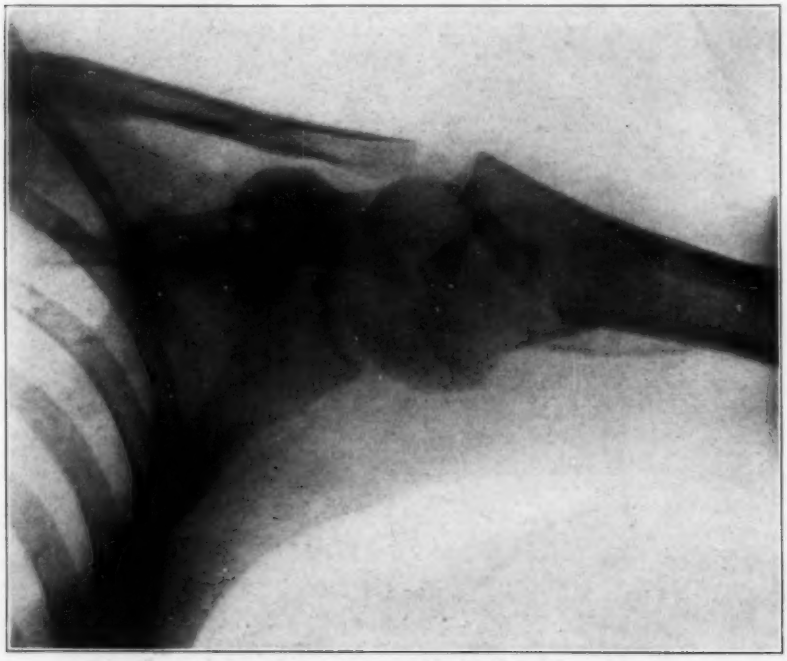

Fig. 6.—Abduction of humerus 90 degrees.

Balkan frame apparatus, the patient slowly assisted into a sitting position in the bed, and a plaster cast applied from the left hand to below the crests of the ilia (fig. 8).

This position was approximately with the humerus abducted 120 degrees with the body, rotated outward 60 degrees and the forearm flexed 90 degrees.

The cast was bivalved horizontally on August 19, so that the upper portion could be removed to permit massage and passive upward and anteroposterior motions. On September 2, the cast was removed, a cravat sling used and passive and active motions permitted to allow full function, which soon followed.

In approximately ten weeks following a fracture dislocation, this man was back on the job with full restoration of function.

The method used has proved satisfactory in this and similar cases. It substantiates the claim made that the soft parts may aid materially

Fig. 7.—Abduction of humerus about 120 degrees. Fracture reduced and perfect alinement. Note intact periosteum between fragments seen below.

Fig. 8.—Patient shown in plaster cast shortly after application in same position as that maintained in the Balkan frame.

in reducing dislocation and getting the parts of fractures in line when by properly directed traction muscle spasm is overcome.

I claim, therefore, that traction in abduction and outward rotation, putting the capsule on the stretch, in early treatment will reduce anterior and inward dislocation of the humeral head and that hyperabduction, carefully guided by a series of x-ray studies, may entirely overcome the nonalinement in this most disabling condition.

In contrast, it may be well to review briefly comments in leading textbooks on the prognosis and treatment of this serious injury.

C. L. Scudder,[2] speaking of fractures, epiphyseal separations and dislocations of the humeral head, says:

"An anaesthetic must be used for examination and diagnosis, but it is even more important, however, that the first retentive dressing be applied with the assistance of an anaesthetic. Traction, counter traction and manipulation will secure coaptation of the fragments. To hold these fragments securely is difficult—to hold any one of these fractures without operative interference may be impossible." He recommends after reduction, a V-shaped axillary pad, coaptation splints, a plaster shoulder-cap and arm and body plaster of paris or crinoline, with arm at the side and forearm across abdomen, thus rotating the lower fragment inward, the malposition of fracture in this region. Further on, he says that "if traction on the humerus and pressure on the loose head in the axilla fails, open incision is to be done, using the McBurney-Porter hook on the upper fragment and if successful, suturing the shaft with absorbable suture or fine silver wire. . . . If it is impossible to reduce the dislocated head or if it is much comminuted, it will be necessary to excise it."

Sir Robert Jones[3] says:

"The upper fragment formed by the articular head is liable to be rotated into any position and dislocated from the glenoid surface . . . A rotated or dislocated intra-articular fragment will very probably require reposition by operation; this is easily done by the anterior deltoid incision, but there is some liability to recurrence of displacement. If this recurrent displacement cannot be prevented by one or two screws driven through the tuberosity, the head of the bone ought to be removed, but resection more extensive than this is to be avoided."

He also recommends the axillary pad to prevent the adduction of the upper end of the lower fragment, and in closed reductions, he straps the inward rotated arm to the body.

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2. Scudder, C. L.: Treatment of Fractures, ed. 3, Philadelphia, W. B. Saunders Company.

3. Jones, Robert: Orthopaedic Surgery of Injuries, London, Oxford University Press, 1921.

According to Jones and Lovett:[4]

"This accident (fracture-dislocation) is very crippling and if all trials at reduction fail, an open operation should be performed and the head reduced, an ivory or bone peg being used, if it is found necessary." The Jones rectangular arm splint is then described. Later they say: "If the fracture is at the surgical neck and the head is dislocated, an open reduction should be effected and a plate or graft used . . . . After all operations for reducing fracture and fracture-dislocation about the shoulder, except for fractures in or near the glenoid cavity, the arm should be kept in the abducted plane." This does not coincide with Jones' recommendation of closed reduction.

F. J. Cotton[5] says:

"Fracture of the surgical neck complicating dislocation does occur and is a most formidable complication . . . . Obviously all our methods of reduction are of little use in this condition; it is possible that enough connection of fragments may be retained to permit of reduction by right angled traction with outward pressure on the head. If such attempt at reduction does not succeed, as it usually does not, there is nothing for it but to operate."

CONCLUSIONS

The temptation is great to avail oneself of the open operative treatment for fractures whereby one can see exactly what is the situation inside and act accordingly.

Here, however, is a condition wherein utilization of the soft parts and not cutting or further traumatization is indicated. They should be used as tractors to overcome dislocation, muscle spasm and non-alinement of fragments at the shoulder joint.

On account of some of the mutilated and impaired functional results seen after manipulative and operative treatment of fracture dislocation of the shoulder, I feel that the method herein advocated should and can be standardized. Further, probably with the exception of the aid of the x-ray, it can be applied in rural communities far away from great medical centers and hospitals.

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4. Jones and Lovett: Orthopaedic Surgery, New York, William Wood & Co., 1923, p. 413.

5. Cotton, F. J.: Dislocations and Joint Fractures, Philadelphia, W. B. Saunders Company, 1924, p. 211.

WOUNDS OF THE HEART

REPORT OF TWO CASES *

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Of penetrating wounds of the thorax, those involving the heart are relatively infrequent. During the past four years, there have been thirty-five cases of gunshot wounds and sixty-one of stab wounds of the chest at the Louisville City Hospital. Only one bullet and one knife encountered the heart; some of the other wounds were very near, striking the pericardium, but none of the patients gave symptoms to warrant operative intervention.

Early history contains records of many cases of injuries to the heart, and in each instance the patient died. Aristotle and Galen believed that heart wounds were always fatal. Hollerius [1] (1498-1562) was the first to oppose this idea; he thought that small wounds which did not bleed excessively could heal. The first authentic record of a healed wound of the heart was by Iodinis Wolf [2] in 1642. He found a scar in the heart of a deer, and believed that the animal had received the wound previously and had recovered. In 1761, Morgagni [3] was of the opinion that a rapid collection of blood in the pericardial cavity could compress the heart and stop its action.

In 1868, Georg Fischer [4] collected from the literature a series of 452 cases of wounds of the heart with a mortality of 90 per cent. The treatment employed at that time was rest, venesection, application of leaches and occasionally, when possible, the passage of a catheter or sound into the wound for evacuation of fluid from the pericardial cavity.

In 1881, Roberts [5] suggested that wounds of the heart might be sutured. Suture of the heart muscle was first performed experimentally on rabbits by Black [6] in 1882. The idea met with much opposition—

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* From the Department of Surgery, University of Louisville School of Medicine.

1. Hollerius, J.: Communis aphorismi allegati, quoted from Beck: Wounds of the Heart, Arch. Surg. **13**:205 (Aug.) 1926.

2. Wolf, Idonis, quoted from Beck.

3. Morgagni, J. B.: De Sedibus et Causis Morborum, Lipsiae, sumptibus Leopoldi Vossii, 1829; quoted from Beck.

4. Fischer, Georg: Die Wunden des Herzens und des Herzbeutels, Arch. f. klin. Chir. **9**:571, 1868; quoted from Beck.

5. Roberts, J. B.: The Surgery of the Pericardium, Am. Anat. & Surg. **4**: 247, 1881.

6. Black: Verhandlungen der deutschen Gesellschaft für Chirurgie, Elften Congress, Berlin, 1882, part 1, p. 108; quoted from Beck.

many prominent surgeons spoke discouragingly concerning the subject. Del Vecchio [7] experimentally incised the hearts of dogs, then sutured the wounds, and the animals recovered. In 1895, he demonstrated these healed wounds to the Eleventh International Medical Congress in Rome, Italy. During the following year, three human hearts were sutured; the first two patients died following the operation, while the third (operated by Rehm [8]) lived and returned to normal life.

In 1920, Tuffier [9] collected 305 cases of sutured wounds of the heart, in 50.4 per cent of which the patients recovered. This probably is a rather high percentage because surgeons are more prone to report their successful cases.

The prognosis depends on the type and extent of the injury. Wounds which penetrate into the cardiac cavity and leave an opening in the heart wall sufficiently large for the escape of much blood are usually fatal in a short time. The clinical picture of the less extensive wounds depend largely on the size of the opening in the pericardium. If there is a fairly large opening providing free escape of blood, the symptoms produced are those of any severe hemorrhage. If the pericardial rent is small and blood collects in the pericardial cavity, compression of the heart results; the arterial pressure is lowered, and the venous pressure is elevated. When the intrapericardial pressure becomes equal to the venous pressure, the heart ceases to function; this condition is called heart tamponade. During the process of compression (or tamponade) cyanosis develops, the pulse grows weaker and more rapid, heart sounds become distant, and respiration is increased.

A surgeon may be called to meet a cardiac emergency at any time; therefore, he should be familiar with the various methods of operative technic, and know how they can best be applied under different conditions. The earlier operations were performed through an opening obtained by the removal of one rib. This, however, gave poor exposure. Later methods include: (1) removal of two or more ribs and costal cartilages subperiosteally; (2) hinged flaps consisting of skin, muscles, ribs and possibly a portion of the sternum; (3) median sternotomy; (4) intercostochondral thoracotomy—that is, two, three, or four costal cartilages are divided near the sternum and the ribs retracted until sufficient exposure is obtained. The last two methods are probably the most satisfactory. Median sternotomy gives the best exposure of the

7. Del Vecchio, S.: Sutura del Cuore, Riforma med. **11**:38, 1895; quoted from Beck.

8. Rehm, L.: Ueber penetrierende Herzwunden und Herznaht, Arch. f. klin. Chir. **55**:315, 1897; quoted from Beck.

9. Tuffier, T.: La chirurgie du coeur, Cinquieme congres de la Societé Internationale de Chirurgie, Paris, July 19-23, 1920; Extrait, Brussels, Haye, 1920; quoted from Beck.

heart and may be used without entering either pleural cavity; however, in most injuries the pleural cavity has already been penetrated.

After the heart has been exposed, there are several methods by which the wound may be found and repaired: 1. The heart may be grasped tightly and forcibly pulled out of the wound, where gradual relaxation will reveal the injury, which can then be sutured. 2. The first impulse would be to plug the bleeding area with the finger. This is difficult to carry out, because the beating organ keeps moving out of the way of the finger. If sufficient pressure is exerted to control the bleeding, the friable muscle is apt to be torn and increase the size of the wound. 3. In 1907, Sauerbruch [10] described a method by which the bleeding could be controlled: The middle finger of the left hand is inserted through the great transverse sinus which is situated below the aorta and pulmonary arteries and above all the veins entering the heart. The fourth and fifth fingers are placed below the veins; the latter can be compressed by bringing the third and fourth fingers closer together, and thus keep all blood from entering the heart. The index finger and thumb are left free to steady the heart while the wound is sutured. This pressure cannot be maintained indefinitely, for the heart tolerates ischemia for only a limited period of time. This period of safety has been worked out on animals and varies from 1½ minutes [8] in the dog to 3¾ minutes [11] in the rabbit. To prolong the ischemia beyond this period of time causes the heart to lose its normal rhythm, which it never regains, or there is subsequent degeneration of the cortical cells. 4. A much simpler procedure was recently developed by Beck and Cutler [12] in doing some experimental work on a method for the surgical relief of mitral stenosis. This method will be given in detail in the first case report.

REPORT OF CASES

CASE 1.—M. P., aged 26 (?), was admitted to Louisville City Hospital at 11 a. m. on Oct. 14, 1926. He had received a gunshot wound in the chest a few minutes before admission, the bullet entering just to the left of the sternum in the fifth intercostal space. There was pain only at the site of injury. Examination did not reveal any pathologic process of the lungs; the heart was normal in size, but the sounds were faint; the pulse was weak, with a rate of 84.

Fluoroscopy was performed. The field of the right lung was slightly hazier than that of the left, but there was no conclusive evidence of fluid or pneumo-

10. Sauerbruch, F.: Die Verwendbarkeit dés Unterdruckverfahrens bei der Herzchirurgie, Arch. f. klin. Chir. **83**:537, 1907; quoted from Beck.

11. Laewen, A., and Sievers, R.: Experimentelle untersuchungen über die chirurgisch wichtigen Abklemmungen des grossen gefässo in der Nähe des Herzens unter besonderer Beruchsichtigung des Verhaltnisse bei der Lungen embolie operation nach Trendelenberg, Deutsche Ztschr. f. Chir. **94**:5, 1908; quoted from Beck.

12. Beck, C. S., and Cutler, E. C.: A Cardiovalvulotome, J. Exper. Med. **40**: 375 (Sept.) 1924.

thorax. The right side of the diaphragm was less active than the left. The heart was slightly globular (water bottle type), the enlargement being especially to the right. Fluid was not seen in the pericardium. The bullet was immediately to the left of the midline, moving with each heart beat and lying close to the anterior wall of the chest. Fluoroscopic examination two hours later did not show any change in the condition.

Operation was performed about three hours after admission by Dr. Victor Hill. A midline incision was made down to and through the sternum from the ensiform cartilage to the attachment of the fourth costal cartilage, where the incision was carried laterally through the left side of the sternum into the fourth interspace. When the left portion of the sternum was retracted, the pericardium was exposed. This was incised and retracted, and there was little fluid in the pericardial cavity. A darkened area could be seen on the anterior portion of the right ventricular wall where the bullet could be felt deep in the heart. A suture was placed in the apex of the heart for traction, according to the technic described by Beck.[13] A suture was then placed in the heart muscle on either side of the bullet. The bullet was removed with a hemostat and a large stream of blood spurted from the wound. The two sutures that had been placed on either side of the wound were then pulled to the opposite side from which they were placed, which thoroughly controlled the bleeding. The wound was then closed with interrupted figure-of-eight silk sutures. (Six of these sutures were used.) The pericardium was closed with interrupted sutures of silk. The sternal reflection was put into place and sutured with kangaroo tendon.

The temperature rose to 103 F. the following day, the pulse steadily increased in rate and became weaker. There were moist râles in the right lung; the left lung seemed clear, but sounds were distant. The patient reacted poorly, and died forty-eight hours following operation.

Postmortem examination showed only a small amount of fluid in the pericardium. The sutures in the heart muscle were removed. The wound extended a distance of 15 mm. into the interventricular septum with a lateral perforation into the cavity of the right ventricle. The left lung was collapsed and airless. There was a slight diminution of crepitation in the right lung.

Case 2.—H. M. A., a colored man, aged 26, was admitted to the Louisville City Hospital on July 16, 1927, with a stab wound 8 cm. in length and extending from the tip of the ensiform cartilage slightly upward and to the left. The patient presented the general picture of shock: The pulse was 160 and weak; the skin was cold and moist; every time he breathed a slushing sound was produced, and considerable blood was sprayed from the wound. He was under the influence of alcohol, which may have contributed toward his semiconscious condition.

An attempt was made to combat shock by subcutaneous infusion of saline and caffeine sodium benzoate, application of external heat and hot coffee by rectum.

After the patient was in the operating room, which was about two and a half hours following the accident, examination showed that the tip of the ensiform cartilage and the cartilages of the fifth, sixth and seventh ribs were cut. Although this opening, which was already made, gave a rather low exposure of the heart, it was fairly satisfactory. The skin and subcutaneous tissues surrounding the injured area were infiltrated with 1 per cent procaine hydrochloride, no other

13. Beck, Claude S.: Wounds of the Heart, Arch. Surg. **13**:205 (Aug.) 1926.

anesthetic being used. The edges of the wound were retracted, and the pulsating heart was seen in a pool of blood.

A suture was placed in the apex of the heart; this was used for traction by holding it in the left hand, while the left index finger examined the heart carefully. There was a longitudinal incision 3 cm. long in the anterior portion of the right ventricular wall, extending to a depth of about 6 mm. Owing to the length of the wound, hemostatic sutures were not used. The index finger held over the wound controlled the bleeding, while interrupted silk sutures were applied to approximate and close the edges of the wound. When this was accomplished bleeding ceased. All the blood was then cleared out of the pericardial cavity; this afforded an excellent view of the wound, which was about 5 mm. from the coronary artery. None of the sutures had pierced or included that structure. The pericardium was closed with interrupted silk sutures, which did not make an air-tight pericardial cavity. There were three openings (each about 15 mm. in diameter) in the left pleural cavity; these were not closed. The wall of the chest was closed air tight. A needle was inserted into the left pleural cavity and about 1,500 cc. of air removed, thus relieving the pneumothorax.

During the first thirty-six hours after the operation the temperature was between 99 and 100 F., the pulse rate between 100 and 126, and the respiration from 58 to 70. The patient's breathing was shallow, and there was some air hunger. He was thirsty, took fluids well, and retained them. His temperature then rose and for four days remained between 101 and 102.4 F. The heart rate averaged around 130 and went up as high as 152. The respiration gradually went down to around 40. Nourishing foods were given, he was digitalized and treatment with small doses of digitalis continued.

July 19: Examination showed pneumonia of the base of the left lung. Pericardial effusion and dulness (extending 6 mm. to the right of the midline) were present, and a pericardial friction rub was heard.

July 22: The left border of the area of cardiac dulness extended to the anterior axillary line and the right border 10 cm. to the right of the midline. There was evidence of fluid in the left pleural cavity at that time. Slight separation of the edges of the wound, with the drainage of a small amount of pus, was noted.

July 24: The pericardial cavity was aspirated through the fourth interspace just to the right of the sternum, and 110 cc. of straw-colored fluid was obtained. The left pleural cavity was aspirated, and only 50 cc. of bloody fluid was obtained. *Staphylococcus aureus* was cultured from the pericardial fluid and from the pus of the wound. The pleural fluid was sterile.

July 26: Two hundred cubic centimeters of fluid was aspirated from the pericardial cavity (fig. 1). *Staphylococcus* was again grown from the fluid. The wound was entirely open, exposing the anterior mediastinum. The pleural cavity was walled off and did not communicate with the wound. A roentgenogram showed bronchopneumonia of the base of the right lung in addition to the large bottle-shaped cardiac shadow and the radiopacity of the base of the left lung.

July 30: The cough increased and was aggravating. Only 50 cc. of fluid was obtained from the pericardial cavity; the fluid was lost, so culture was not obtained.

August 4: One hundred and fifty cubic centimeters of sterile fluid was aspirated from the pericardial cavity. The patient seemed stronger, but the temperature remained around 101 F.; the pulse rate was 140, and the respiration, 50.

August 13: One hundred and fifty cubic centimeters of sterile fluid was aspirated from the pericardial cavity. The temperature was 100 F.; the pulse rate, 120, and respiration, 40. The patient's condition was much better, although he continued to have an irritating cough. The chest wound was clean and granulating.

August 20: The patient was so well that he was allowed to sit up for a short while. The temperature had been normal for six days previously.

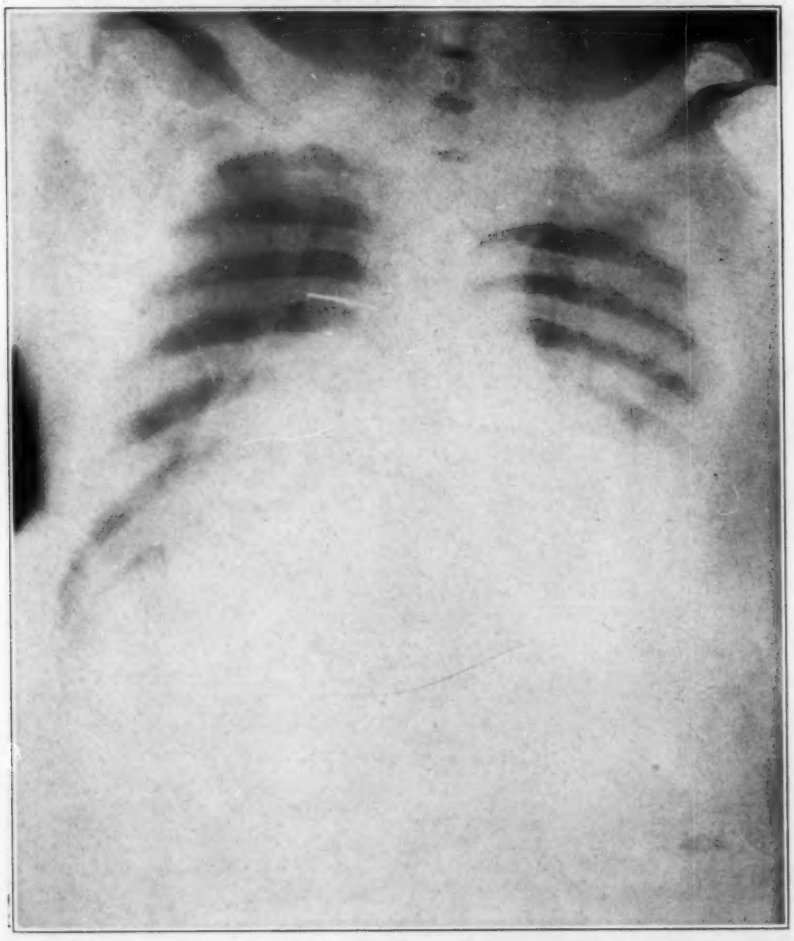

Fig. 1.—Roentgenogram taken July 26, 1927, showing enlargement of the cardiac shadow, bronchopneumonia at the base of the right lung and radiopacity of the base of the left lung.

August 22: Dyspnea and generalized anasarca was found on examination (fig. 2).

During the next six weeks, general treatment was given for cardiac decompensation, the etiology of which was considered to be myocardial injury, adhesive pericarditis or both. The treatment in general consisted of the administration of

digitalis, limitation of the fluid intake and methods favoring the excretion of fluids. Merbaphen was given repeatedly at from two to five day intervals; a large output of urine followed the use of this drug, and there was a noticeable reduction of edema.

Edema was not noticeable after October 7. The wound had healed. The fluid intake was gradually increased, and was well tolerated. Antisyphilitic treatment was started.

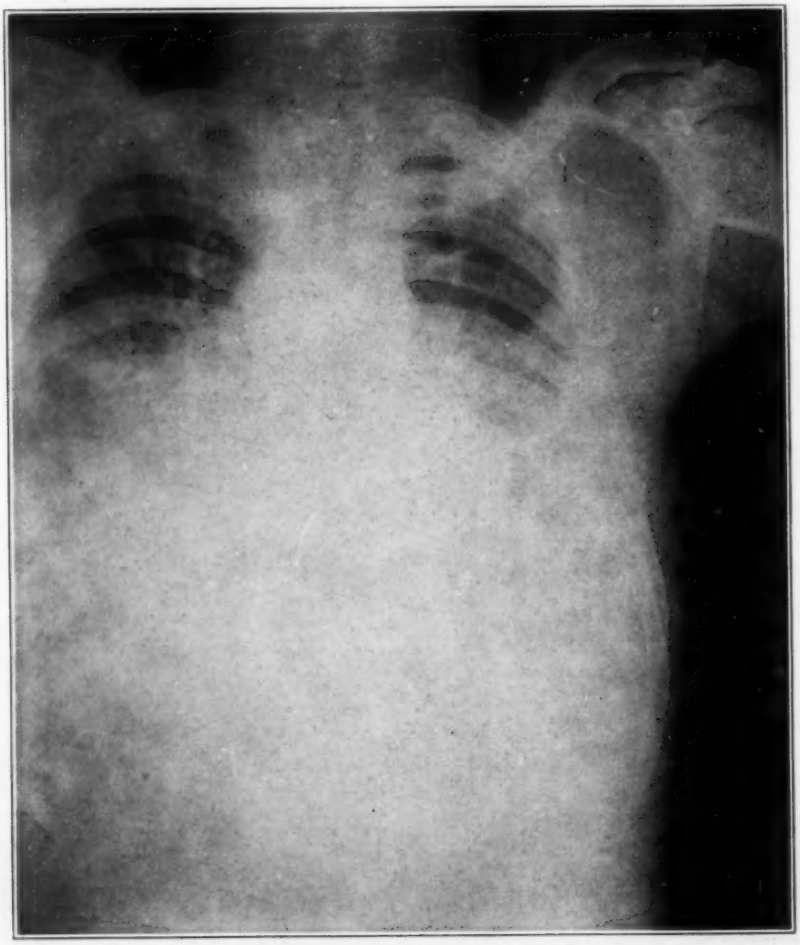

Fig. 2.—Roentgenogram taken Aug. 22, 1927, showing fluid in both pleural cavities.

October 20: The patient was allowed out of bed. He would not take exercise gradually, but walked about at will the second day he was up; he did not suffer any ill effects, and continued to go about as a normal person.

October 30: He was discharged from the hospital in apparently good physical condition; the fields of both lungs were clear, and his heart was 12 cm. in its greatest diameter (fig. 3).

November 9: He was seen for the last time, and seemed normal.

COMMENT

The method of handling the heart by the traction suture in the apex enables the procedure to be carried out with much less trauma than by any other method now known. The placing of sutures alongside the wound to control hemorrhage prevents soiling of the operative field.

Many of the early operators left a drain in the pericardial cavity. Adhesive pericarditis almost invariably followed these cases. In most of

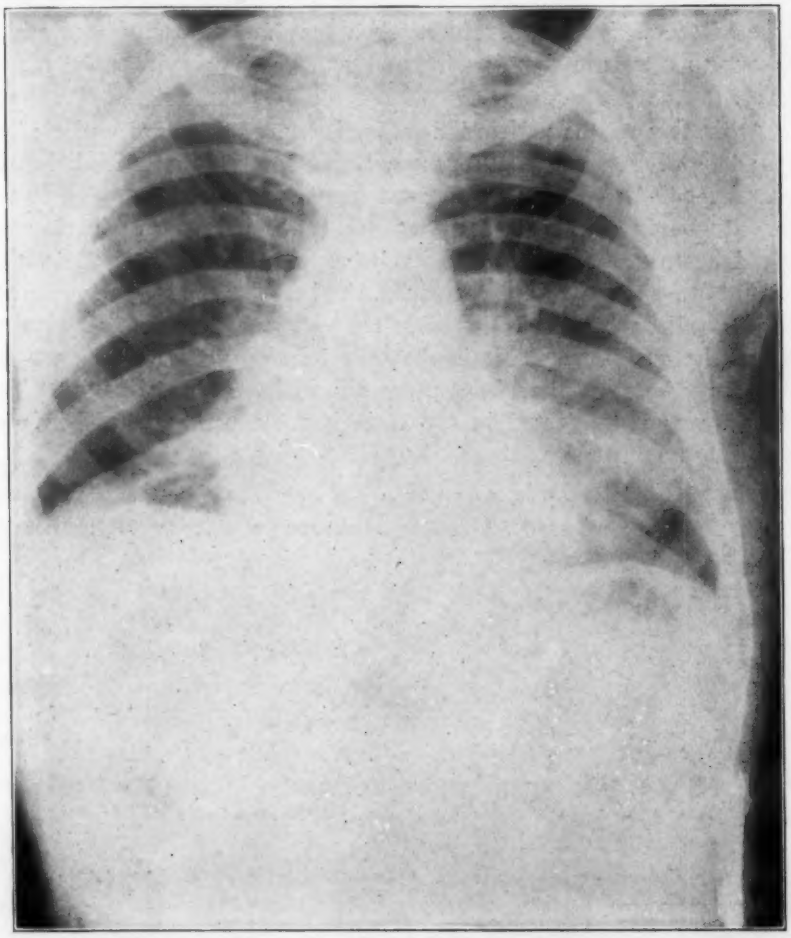

Fig. 3.—Roentgenogram of patient just before leaving hospital on Oct. 30, 1927, showing a return to almost normal.

those cases in which drainage was not used, pericardial effusion developed. These observations led to some exerimental work by Beck and Moore,[14] who found that mild trauma and the application of a chemical

14. Beck, C. S., and Moore, R. L.: The Significance of the Pericardium in Relation to Surgery of the Heart, Arch. Surg. **11**:550 (Oct.) 1925.

irritant to the pericardium cause either pericardial effusion or adhesions or both. Small amounts of blood would be injected into the pericardial cavity, the blood would be absorbed, and effusion or adhesions did not develop. These experiments bring out forcibly that it is most important to produce as little trauma as possible. The sponging of the blood from the cavity with gauze produces trauma and suction should be substituted when possible.

The associated pneumothorax may be a factor in lowering the resistance of the patient. Therefore, when feasible, it would seem advantageous to close the thorax tight and remove the air from the pleural cavity.

Aspiration of the pericardial cavity is a procedure not entirely free from danger. A coronary artery may be punctured, the pericardial cavity becoming filled with blood, and fatal tamponade result. Owing to this possibility—such an accident has occurred—suppurative pericarditis should be treated by open incision when the condition of the patient warrants that procedure. Ordinarily, the best location for the insertion of the needle, when aspiration is to be performed, is just below to the left of the ensiform. This is the only location where the pericardial cavity may be entered without penetration of the pleural cavity. This location was not selected in my case, because the obviously infected wound was at that site.

NOTE.—The patient returned to the hospital on May 1, 1928, in perfect health. He said that he had not had any cardiac embarrassment since leaving the hospital.

SPONTANEOUS DISLOCATION AND DESTRUCTION OF TENDON OF LONG HEAD OF BICEPS BRACHII

FIFTY-NINE INSTANCES *

A. W. MEYER

PALO ALTO, CALIF.

In bringing these two conditions together in the title, I am not assuming that they necessarily are always associated and that every case of destruction of this tendon necessarily was first one of dislocation. In most cases, however, dislocation of the tendon probably leads to greater wear, but, as reported previously,[1] tendons still lying in a normal position and in otherwise normal shoulder joints may show considerable wear.

Since the biceps as well as other muscles may be congenitally absent, it follows that the tendon of either head may also be absent congenitally, but it is improbable that all the statements in the literature on this matter can be taken at their face value. I never have seen a clear case of congenital absence of the long head since I have understood the condition better, and few of those reported in the literature, including my own,[2] are absolutely convincing in view of the facts I have subsequently revealed. Such cases as those spoken of by Lauth [3] and Lecco [4] seem unequivocal, but what is needed is examination of the tendon in a large series of the new-born.

I do not believe that the prominence of the supraglenoid tubercle is a reliable criterion for deciding whether the tendon of the long head is congenitally absent; for, as shown in figure 1, the tendon of the long head frequently merges so completely with the ligamentous, glenoidal lip as to be indistinguishable and also inseparable from it. One could therefore expect great variations in the prominence of the tubercle for this reason alone. In such cases as that reported by Craig,[5] the downward extension of the so-called transverse humeral ligament suggests

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* From the Department of Anatomy, Stanford University.

* This does not include a number of instances in the twenty-five bodies under dissection this year.

1. Meyer, A. W.: Further Evidences of Attrition in the Human Body, Am. J. Anat. **34**:241, 1924.

2. Meyer, A. W.: Absence of the Tendon of the Long Head of the Biceps, J. Anat. and Phys. **48**:...., 1913-1914.

3. Lauth, Ernest Alexandre: Nouveau manuel de l'anatomiste, Paris, 1829.

4. Lecco, T. M.: Ein Fall von vollständigen Fehlen des langen Kopfes des M. biceps brachii und die damit in Zusammenhang stehenden Veränderungen an Knochen und Gelenktheilen, Anat. Anz. 1907, vol. 30.

5. Craig, Joseph D.: Anomaly of the Long Tendon of the Biceps Muscle of the Arm, New York M. Record, 1899, vol. 56.

that the tendon of the long head was not congenitally absent, but that it had secured a secondary attachment after division. Since neither my associates nor I have seen a single convincing case of congenital absence of the tendon of the long head in adults, in more than a thousand arms, it is clear that this condition is rare, although absence of the long head is common. Indeed, the high incidence of the latter itself compels another interpretation than that of developmental anomaly alone.

In previous publications,[6] I have tried to convey the idea that partial or complete destruction of the articular portion of this tendon is relatively common and not unique, as King and Holmes [7] seem to imply.

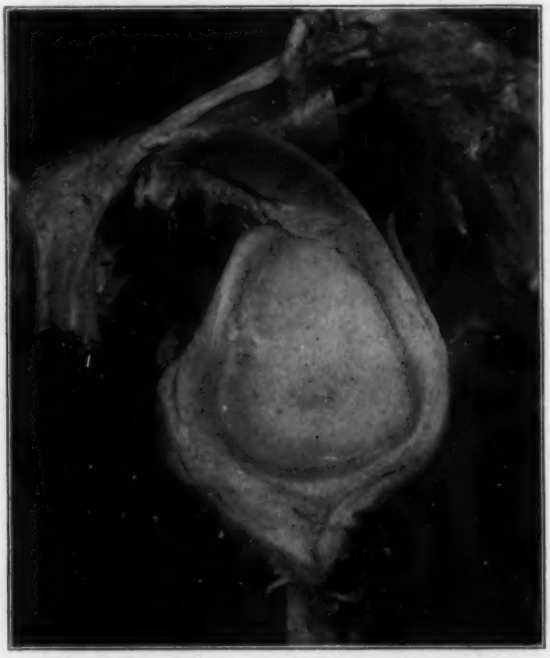

Fig. 1.—Left scapula, illustrating the apparently total fusion of the long head of the biceps with the dorsal portion of the ligamentous lip.

I have thirty-nine examples of dislocation and twenty of complete absence of the articular portion of this tendon in my possession at present. Although only six of the cases of dislocation are maximal, cases in which the tendon had passed completely over the lesser tuberosity,

6. Meyer, A. W.: Unrecognized Occupational Destruction of the Tendon of the Long Head of the Biceps Brachii, Arch. Surg. **2**:130 (Jan.) 1921; Further Observations Upon Use-Destruction in Joints, J. Bone & Joint Surg. **4**:491,1922; Spontaneous Dislocation of the Tendon of the Long Head of the Biceps Brachii, Arch. Surg. **13**:109 (July) 1926; footnote 1.

7. King, Jennings M., and Holmes, George W.: The Diagnosis and Treatment of Painful Shoulders, J. A. M. A. **89**:1956 (Dec. 3) 1927.

most all of the rest are wholly unequivocal to any one familiar with the condition. In all the cases of absence of the articular portion, the tendon had obtained a secondary attachment to the floor or sides of the sulcus or to the humeral diaphysis directly distal to the lesser tuberosity, as represented in figures 4, 5 and 6.[8]

Although complete absence of the intra-articular portion (destruction) occurred more frequently on the right than on the left, the opposite is true of dislocation; however, since both series still are small, not much importance can be attached to conclusions drawn from them. Thirteen cases of complete destruction occurred on the right and seven on the left, and seventeen cases of dislocation occurred on the right and twenty-two on the left. Both sexes are represented, and in four cases the dislocation was bilateral. As I did not preserve all cases of destruction of the tendon because they are so common, and did not discover such a possibility as spontaneous dislocation until a few years ago, it is probable that I will be able to extend these observations considerably during the next few years. Many cases probably were overlooked.

I realize that the word spontaneous is out of place here as elsewhere, but I am using it to distinguish these cases of dislocation from those resulting from sudden trauma due to exertion or to external causes. Some trauma undoubtedly is associated with these cases of gradual occupational dislocation, but trauma, in the ordinary sense, is not a factor. Nor do I wish to rule out chronic arthritis. Evidences of its presence are unmistakable in some cases, but there is no reason to suppose that people afflicted with chronic arthritis thereby become immune to dislocation. They would, of course, be more likely to suffer it because of the changes in bone and capsule associated with this disease.

In some cases in which the divided tendon obtained a secondary attachment to the diaphysis distal to the lesser tuberosity, it may have been dislocated and have played on the tuberosity before it was divided; hence it is possible that most of these cases should really be included among cases of dislocation. It is not improbable that this also is true of the cases in which the tendon is attached to the floor of the sulcus in the region of the lesser tuberosity, for it is not necessary for the tendon to be exposed to considerable wear from contact with the relatively rough face of the lesser tuberosity, in order to be weakened by wear.

PARTIAL DISLOCATION

Since the observations published in 1926, I have found that partial dislocation of this tendon is a far commoner condition in bodies in our dissecting room than I had surmised. As stated at that time, I did not recognize the cases of beginning dislocation at first, and in 1924, I

8. Meyer (footnote 6, first reference).

published a photograph (fig. 7) of a relatively early case without interpreting it as such. The paired humeri represented in figure 2 illustrate a still earlier stage in a case of dislocation. The condition of the sulcus on the left humerus represents an earlier stage than that on the right, for in the latter the tendon had already left the proximal part of the sulcus and had come to play on its anterior wall and crest and on the lesser tuberosity. In all the cases of dislocation I have seen, the tendon still occupied the sulcus in the region distal to the tuberosities. As the sulcus is shallow here, it is difficult to see how it would be possible to determine the absence of the tendon by palpation, and those

Fig. 2.—Paired humeri, illustrating instances of beginning dislocation. The widening of the proximal portions of the sulci should be noted.

who have claimed that they have found the sulcus empty in the living, must of necessity have determined this by palpation through skin, subcutaneous tissues, fasciae and the thick layer of muscles (pectoralis major and deltoid) overlying the more proximal portion of the sulcus, which is the only region in which dislocation is known to occur spontaneously; that is, gradually from use.

Since the bodies used in the anatomic laboratory at Stanford University are not drawn from an unusual source but represent the classes and occupations usually represented in this material, I see no reason why the incidence of bicipital dislocation in this laboratory should be

unusual. With a few exceptions, practically all the bodies are of persons who belonged to the working classes. The great majority were Caucasian, only a few having been negroes, Chinese, Mexican and Hindus.

The greater frequency of absence of the intra-articular portion of the tendon on the right side may be due to right-handedness, and the somewhat larger frequency of dislocation on the left may be due to the fact that greater tension is put on the tendon and the capsular attachment on this side, in occupations that require shoveling and pitching with a fork, for the left hand acts as fulcrum and the left humerus passes into marked lateral rotation and abduction in every such movement.

ANATOMIC FEATURES

In considering the anatomy of the humeroscapular articulation, one is impressed with the fact that the normal conditions promote dislocation of the tendon of the long head. Until the arm is abducted somewhat, the tendon curves forward, encircling the slippery and sloping, rounded surface of the upper, anterior portion of the head of the humerus. As tension on the tendon is increased, it tends to displace the latter backward and downward. This does not imply, however, that a gap, such as appears in roentgenograms of the shoulder with the arm at rest, exists between the head of the humerus and the acromion. Muscle tone alone always must keep these in contact. The large gap seen in roentgenograms of the shoulder is due, of course, to the presence of the articular cartilage and capsule and the tendon of the supraspinatus, and is magnified because the lower surface of the acromion and the cartilage on and the underlying compacta of the humeral head, are curved bodies. Since the humero-acromial interval is decreased decidedly in all cases of destruction of the upper portion of the capsule and the tendon of the supraspinatus and also by wear on the under surface of the acromion and on the articular cartilage of the humeral head, this decrease should be evident in roentgenograms.

Although the articular cartilage on the humerus is sometimes grooved slightly by the tendon in the region adjacent to the tuberosities, this sulcus is too shallow to have much, if any, influence in maintaining the normal position of the tendon, and only that portion which lies between the tuberosities in adduction, is properly accommodated by the normal sulcus. The tendon usually widens and flattens decidedly as the supraglenoid tubercle is approached, where it sometimes is three, or more, times as wide as in the intertuberosital portion, when the arm is at rest. Since anatomists now speak of the entire bicepital sulcus as intertubercular, I am using the word intertuberosital to designate the portion between the greater and lesser tuberosities.

The relation of the tendon to the head of the humerus, and to the floor of the intertubercular sulcus, necessarily changes as the position of the arm changes, for both shift beneath it. When the arm is in slight lateral rotation, the under surface of the tendon lies fully on the floor of the sulcus; but as lateral rotation is increased, its anterior margin is forced against the anterior wall of the sulcus, especially that portion formed by the lesser tuberosity and the capsular attachment proximal to it. In medial rotation, on the other hand, the anterior wall of the sulcus and the adjacent capsular attachment become the surfaces which the tendon uses as a trochlea, while its dorsal margin is forced against the dorsal wall of the sulcus.

It is evident that the tension exerted on the capsular attachment proximal and superior to the lesser tuberosity varies with the position of the arm and the disproportion in width between the tendon and the sulcus, as well as with the force exerted by the muscle on the tendon. It seems probable that the greatest tension is exerted on this portion of the capsule which is the first and chief obstacle to forward dislocation especially in the position of extreme lateral rotation during partial abduction. This is the position frequently reached in occupations that require shoveling. In extreme abduction the relations would be entirely different, and the tendency to dislocation would probably be slight because of the approximation of the lesser tuberosity to the supraglenoid tubercle.

The tendency to dislocation of this tendon, which exists in all normal shoulder joints, is opposed primarily by the attachment of the articular capsule in the region proximal to the lesser tuberosity. Contrary to the usual statement, I do not believe that the so-called transverse humeral ligament is an important factor for preventing dislocations, for I have seen cases in which the tendon was entirely normal in position, although this ligament was extremely weak or absent. Moreover, after observing the condition of the intertubercular portion of the capsule, I fully concur in the omission of a name for this so-called ligament from the Basle nomenclature. Transverse fibers bridging the sulcus, which could be designated properly as a ligament, seldom are present, and when this is the case, they usually are located farther distally than the intertubercular region, in which the transverse humeral ligament of Brodie is supposed to occur.

I have seen only one case of complete ossification of the intertubercular portion of the capsule and another in which ossification was partial. In the former, the tendon passed through a bony canal about 2 cc. long as in the mole, armadillo and musk ox, and in the latter case it was covered medially by an exostoses. It is such rare instances as these that may have phylogenetic significance and hence justify the term transverse humeral ligament, but careful developmental and statistical studies are necessary to establish this.

The nature of the anterior wall of the sulcus necessarily is an important factor in dislocation. This applies especially to the presence or absence of the bony ridge shown in figure 3, which I have called the supratubercular ridge. Although this name might imply that it is related to either tuberosity, I have never seen it related to the greater. It extends obliquely forward and downward from the region of the articular cartilage to the upper and dorsal portion of the lesser tuberosity. As the surface of the distal extremity of this ridge is on a level with the surface of the latter, and since the tendon plays on it

Fig. 3.—Portion of a right humerus with a supratubercular ridge marked X.

much as the tendon of the peroneus longus plays on the trochlea of the cuboid, its rôle in dislocation is plainly evident. In a study made by Cilley (unpublished) it was present in 17.5 per cent of 200 humeri.

In all normal joints there are three, and there may be six, factors which favor dislocation of this tendon: (*1*) the normal course of the intracapsular portion of the tendon and its relation to the humeral head, (*2*) the much greater width of the proximal portion of the tendon, (*3*) the fact that the anterior wall of the sulcus which is formed by the lesser tuberosity normally acts as a trochlea for the tendon in the usual position of medial rotation, (*4*) a supratubercular ridge may be

present, (5) the capsular attachment may be weakened by intracapsular bursae and (6) the capsular attachment to the anatomic neck in the region proximal to the lesser tuberosity may be restricted.

INTRAMURAL BURSAE

So far as I know, the presence of bursae in the capsular attachment in this region has not heretofore been reported; yet, not infrequently, one or two small intracapsular supraperiosteal bursae are present. The presence of these bursae in this region of capsular attachment results in the weakening of it. I am not certain as to the genesis of these bursae, but I do not believe that they are congenital, but rather that they result from the play of the tendon of the long head against and on this portion of the capsular attachment, especially during marked lateral rotation. Not infrequently the bursa of the subscapularis also extends far up under the tendon of this muscle, and it may even communicate with the sheath of the long head in the intertubercular sulcus. This may also be the case with the intracapsular bursa or bursae which form a gap bridged by the capsule or the tendon of the subscapularis, as the case may be. Such defects in the capsular attachment must greatly facilitate dislocation of the tendon, for they weaken the primary obstacle against its inception. Although the articular capsule and the tendon of the subscapularis always fuse intimately, it is only the capsular attachment which is an obstacle to dislocation, for the dislocated tendon of the long head does not detach the tendon of the subscapularis but comes to lie on it in far advanced cases, as illustrated in the figures referred to. If the capsular attachment to the bone in this region is maintained and stretches, the tendon of the long head comes to lie in a capsular sling, as shown in figure 4. This necessarily protects it against attrition on the face of the lesser tuberosity. Under these circumstances, it may become dislocated without showing much or even any wear from contact with the lesser tuberosity, and such cases are not rare.

Since in all the cases of detachment of the tendon of the subscapularis over practically the entire lesser tuberosity some arthritic reactions are shown, it seems to me that the gradual dislocation of this tendon probably results from disease, although mechanical factors are involved. In some cases the tendon of the subscapularis retains an attachment anterior and distal (below) to the lesser tuberosity only, and looks as though it had undergone a slow migration from the region of its wide normal attachment. It must not be overlooked, however, that abnormalities in the attachment of this tendon may also be concerned in this matter.

Spontaneous dislocation of the tendon of the long head of the biceps may occur without the presence of arthritis, a supratubercular

ridge or bursae in the region of the capsular attachment, and also without the restriction of this attachment to the upper margin of the lesser tuberosity. Although it must remain a matter of opinion as to just what constitutes beginning dislocation, I think it conservative to say that approximately 4 per cent of the bodies and 8 per cent of the

Fig. 4.—A right humeroscapular articulation with the tendon reflected anteriorly to reveal its bed. The proximal portion of the intertubercular sulcus is seen to the left of the new bed of the tendon. This tendon, which lies in a capsular sling, had passed practically over the lesser tuberosity but still occupied the sulcus distal to the intertuberosital region.

tendons illustrate some stage of this process. A sufficient number of these cases present no evidences of pathologic conditions to rule out arthritis as a primary or sole cause.

Not infrequently the floor of the intertubercular sulcus slopes decidedly toward the greater tuberosity in its upper extent, thus tending to throw the tendon against the dorsal wall of the sulcus and facilitating wear on this margin, as shown in figure 5. The first instances of this kind which I saw led me to think that the wear of the tendon here must have resulted mainly from its being caught between the greater tuberosity and the acromion, after a defect had been formed in the articular capsule; but the subsequent finding of worn tendons in joints with intact, normal capsules made this interpretation untenable. I have also found that the tendon can be dislocated without previous detachment of the articular capsule, for the latter may merely stretch in the region of the lesser tuberosity instead of becoming detached.

Fig. 5.—Three left bicipital tendons from otherwise wholly normal joints, showing wear along the dorsal margins. The reflection of the tendon sheath is clearly shown in number 1. All the tendons were cut close to their origin.

The fraying of its dorsal margin manifestly is produced by contact with the dorsal wall of the sulcus and is especially marked in tendons which are much wider in their proximal portions. Fraying of this edge may extend to within 0.5 cc. of the supraglenoidal attachment. I do not know whether or not wear on this margin, under otherwise normal conditions, would ever be sufficient to divide the tendon, but I am inclined to doubt it. It could, however, not fail to weaken it and thus facilitate its rupture.

In some cases in which the tendon had ruptured some distance from its place of origin, the proximal fragment could be recognized, although it had become adherent to the inner surface of the capsule. Often it

could not be traced without difficulty, and in some cases not at all. Its fusion with the capsule may be due to traumatism, and its atrophy to disuse.

Although the incidence of spontaneous dislocation—and destruction—in the general population undoubtedly is far less than in the material in the dissecting room, it must be exceedingly common in persons who work in lumber and construction camps and in mines. Although the condition here described is undoubtedly chronic, it must be more or less distressing, even if not disabling, until maximal conditions are reached. In cases in which the intertubercular sulcus has not undergone secondary changes which have resulted in narrowing or roughening of it, relocation of the tendon, if not too badly worn, would seem justifiable if the tendon can be secured against recurring dislocation. Little could

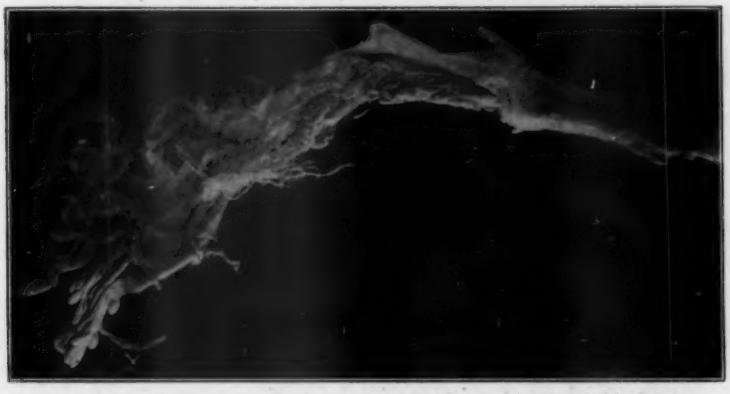

Fig. 6.—Right tendon of the long head photographed from beneath, showing maximal wear. This tendon was attached only by a fine strand marked x, in the normal region and shows wear all the way down to the region of the reflection of its synovial sheath.

be accomplished, however, by relocating such a tendon as shown in figure 6, though it could be fastened securely to the diaphysis so as to restore normal tension on it and thus prevent a certain amount of atrophy of the lateral portion of the muscular belly of the biceps.

Except in early stages, I doubt, however, whether much can be gained in muscular power by reattachment of the tendon of the supraspinatus, so commonly divided by attrition, or displaced dorsally in cases of attrition. To reintroduce what remains between the bony surfaces involved might merely reestablish former disabilities, for the shortened tendon and muscle would have to be stretched considerably and would again be subjected to disastrous wear unless the patient changed his occupation.

In view of the undoubted commonness of destruction and dislocation of the tendon of the long head of the biceps, it is especially regrettable that clinical histories are not available. This is no one's fault, of course, for it is unlikely that any of the persons so affected sought relief from conditions of long standing by seeking medical care during later years. Moreover, the medical staffs of relief homes, almshouses and insane asylums are too small to permit much attention being given to slight disabilities or aches felt by the aged. However, although they remained unknown until recently, the conditions nevertheless are of peculiar scientific interest and also of great practical importance, for they undoubtedly were sustained during the most active years of the person's life.

SUPERNUMERARY CAPSULAR HEADS OF THE BICEPS

While studying the humeroscapular articulation in specimens at hand, I found three cases in which the biceps had a third tendinous or capsular head. In two specimens with unruptured tendons, these tendinous cords arose from the tendon of the long head in the region of the lesser tuberosity and extended to the upper anterior and inner surface of the articular capsule. These cords fused completely with the inner surface of the capsule and gradually thinned out as they continued toward the region of the supraglenoid tubercle. They are, however, entirely distinct from the long head and have exactly the same structure, although the largest is only half as thick as the entire tendon. The presence of such a capsular head would tend to prevent too great a retraction of the long head when the latter is divided, and thus would facilitate its secondary attachment in the region of the lesser tuberosity. It also seems that the traction exerted on the capsule by it might cause discomfort if not pain, especially in cases in which the tendon of the long head had stretched because of weakening from attrition.

These capsular heads of the biceps mentioned by Moser, Henle and Welcker, left the posterior and upper surface of the main tendon in the region of reflection of the synovial sheath. The largest lay adjacent to the main tendon, but one of the smaller occupied a separate, superimposed compartment. The former could be traced along the under surface of the capsule as far as the ligamentous lip of the glenoid cavity, in the region of the supraglenoid tubercle, but they were an unmistakable portion of the tendon of the long head. In the third case the supernumerary tendon lay outside of the synovial sheath of the tendon of the long head, but joined the capsule in the same region as the others. It widened out considerably distally and apparently joined the tendon of the pectoralis major. Too much had been removed in dissection to enable me to follow it further distally, but since it was attached throughout its extent along

its dorsal border in the region of the intertubercular sulcus and was fused so completely with the fascia proximal to the tendon of the pectoralis major, it is unlikely that it had a muscular component distinct from the biceps, and even if it did these supernumerary tendinous heads are totally different than the well known third muscular head of the biceps so commonly present.

None of these capsular heads could have developed from a portion of the synovial sheath which became strengthened through tension made on it, and since the tendon of the long head develops wholly outside of the articular capsule, and completes its migration inward as late as the third month of prenatal life, it need not surprise one that a portion of it may become stranded and fuse with the articular capsule through which it must pass. Indeed, it seems as though the entire tendon should some time be found in an extracapsular position. As far as I know this has not been observed. The tendinous component which arose from the articular capsule and ended near the tendon of the pectoralis major, if not in it, seems comparable to the glenobrachial muscle of Gruber; but since the other capsular heads here reported are supernumerary, they differ from cases in which all of the tendon of the long head arises from the joint capsule. It seems probable to me that anomalies such as these well may give rise to symptoms and thus make diagnosis of obscure conditions of the shoulder still more difficult. Any one especially interested in these heads will find them considered by Welcker.[9]

CONCLUSIONS

1. Uncomplicated, partial or complete, spontaneous forward dislocation of the supratuberosital and intertuberosital portions of the tendon of the long head of the biceps, on to or over the lesser tuberosity as far as the tendon of the subscapularis, is common in laborers past middle life.

2. This dislocation may be due to gradual stretching, detachment or destruction of the capsule proximal to and in the region of the lesser tuberosity.

3. Wear of the tendon along its dorsal margin may occur in otherwise normal joints, but wear throughout its width probably is frequently due to contact with the lesser tuberosity in cases of dislocation, or with the greater tuberosity and acromion in the course of destruction of the dorsal portion of the articular capsule, unaccompanied by dislocation.

4. After division by attrition, the tendon usually obtains a secondary attachment to the humeral diaphysis immediately distal to the lesser

9. Welcker, Hermann: II. Ueber Muskelvarietäten, Ztschr. f. Anat. u. Entwgsch., 1876, vol. 1.

tuberosity, or to the floor of the sulcus in this region, or still more infrequently to both walls of the sulcus, a portion of the frayed tendon being drawn to each side by the tendon sheath in the region of reflection of the latter.

5. In some cases in which the tendon is divided near the supraglenoid attachment, it retracts completely before it obtains a secondary attachment, with great lengthening of the extracapsular portion—even up to 100 per cent—and marked atrophy of the lateral belly of the muscle and resultant asymmetry.

INTRA-INTESTINAL PRESSURE IN OBSTRUCTION*

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The presence of a toxic substance within the lumen of the bowel in cases of intestinal obstruction has long been recognized, but the process by which it enters the system, if indeed it is absorbed, is a problem that is as yet unsolved. The observations of Stone and Firor,[1] in 1924, suggested the possibility that the changed intra-intestinal pressure might alter the intestinal permeability. Without attempting to investigate the latter problem, it appeared that a study of the degree of pressure within the lumen of the bowel during obstruction might be undertaken with profit.

Before attempting to determine the abnormal conditions, it was necessary to find out the normal intra-intestinal pressure and the relationship of the intra-abdominal pressure to that within the intestine. The physiology of the gastro-intestinal tract from a mechanical point of view reveals an extremely complicated conducting system. The stomach, serving as a reservoir, must receive and readily accommodate the food that arrives often in large volume. Function requires that this mass be stored with little change in the intragastric tension, or else we should experience constant dysphagia, lack of appetite, gastric uneasiness and pain. Sherrington[2] confirmed this and found that the introgastric pressure in an etherized dog over a period of twenty minutes was from 4 to 5 cm. of water. The stomach at this time contained portions of a previous meal, but after 400 cc. of water was put into the stomach through a cannula, the pressure rose only to 6 cm. of water and twenty minutes later had fallen to 4 cm. On the other hand, the small intestine has not such great volumes to handle, but receives the products of the antrum pylori in smaller amounts and over a longer period of time. Such being the case, we would expect an intra-intestinal pressure less than that within the stomach. In the experiment already mentioned,

* From the Department of Surgery, Hunterian Laboratory, The Johns Hopkins University.

* Aided by the Hartley Fund.

1. Stone, H. B., and Firor, W. M.: Tr. South. Surg. & Gynec. Ass. **87**:173, 1924.

2. Sherrington, C. E.: Brain **38**:191, 1915.

Sherrington found that in the jejunum 40 cm. below the pylorus, the intrajejunal pressure varied between 2 and 4 cm. of water.

By a slightly different procedure, we have confirmed these observations of the normal intra-intestinal pressure in dogs, and at the same time we have noted something of the relationship of the intra-abdominal pressure to that within the intestine in which obstruction has been produced. We share the view of Emerson [3] that a slight positive pressure normally exists within the abdomen, that it is dependent for the most part on the tonus of the abdominal musculature and that normal alterations are due chiefly to the contractions of the diaphragm, change in tonus of the abdominal wall and variations in the bulk of the abdominal viscera.

During the course of this study, we have made observations on the intra-intestinal pressure in some eighteen small dogs (from 8 to 12 pounds [3.6 to 5.4 Kg.]) in each of which simple intestinal obstruction had been artificially produced. The obstructions were made for the most part at two levels, one from about 4 to 6 inches (10.1-15.2 cm.) below the duodenojejunal junction, the other an equal distance above the ileocecal valve. In four animals, the obstruction was made in about the middle of the small intestine. Isolated intestinal loops were made in four other animals, and the pressure recorded within the loop.

TECHNIC

Except when otherwise stated, all animals were given ¼ grain (0.016 Gm.) of morphine and ether anesthesia before operation. As strict aseptic precautions were taken as possible. In each case the bowel was doubly clamped and cut across. The lower end was turned in by the Parker-Kerr method and reinforced by a double row of mattress sutures. The clamp was removed from the proximal portion of the bowel and its lumen opened into which the apparatus used was then placed. It consisted of a brass tube about 2 inches (5.08 cm.) in length, with an internal diameter of 3 mm. and a threaded external surface. A concave metal disk about 1.5 cm. in diameter was fixed at one end, and a rubber washer was placed over the disk. A small incision was made in the wall of the bowel about 2 inches proximal to the site of section. The free end of the tube was placed within the lumen of the intestine and brought out through the small incision. The disk fixed one end of the tube within the intestine. The end of the bowel was closed in the same manner as the distal portion. A piece of omentum was then placed over the tube, another rubber washer applied and a second concave metal disk screwed down over the whole, fixing the omentum and the wall of the bowel, not too tightly, between the two rubber washers. A short rubber tube was then attached to the end of the metal tube and brought out through the abdominal wall by a separate stab incision. A small metal clamp closed the rubber tube.

At varying intervals after the operation until the death of the animal, readings were made of the degree of intra-intestinal pressure, and graphic records of the

3. Emerson, Haven: Intra-Abdominal Pressures, Arch. Int. Med. **7**:754 (June 15) 1911.

motility of the bowel were obtained by a recording mercury manometer. Through a T-tube, a water manometer, 3 mm. in diameter, was attached in order to measure the lesser changes in pressure.

RESULTS

For the first twenty-four hours following operation, there was only a slight rise in the intra-intestinal pressure as a general rule, though in one case a considerable rise occurred within six hours. Usually

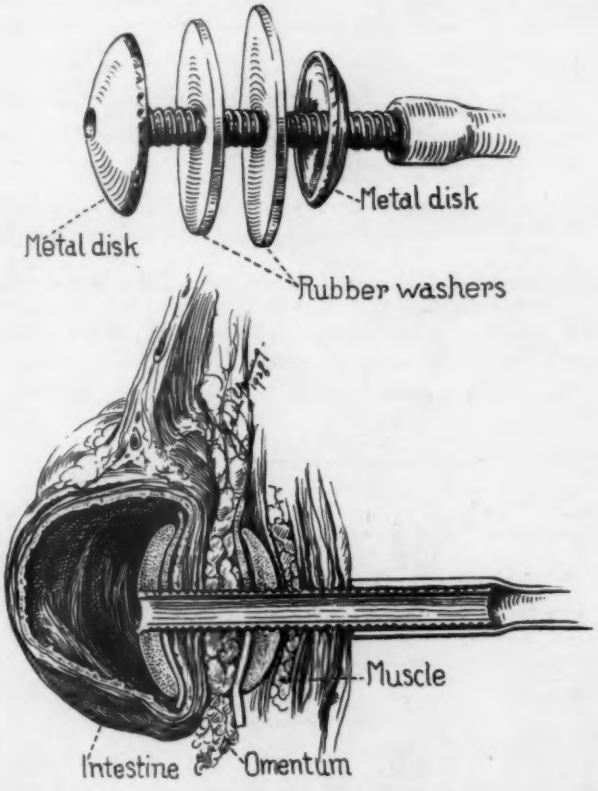

Fig. 1.—Drawing of apparatus described in technic is shown above; that of a cross-section of the apparatus in situ is shown below.

within twenty-four hours irregular peristaltic waves that reached a maximum of from 12 to 16 cm. of water were recorded. Between these there were inactive intervals during which the sustained pressure showed a gradual rise to 6 or 8 cm. As compared with the later observations, the bowel showed less activity during this early period probably owing, in the most part, to the operative trauma, the anesthesia and other factors. So far as the anesthesia was concerned, however, dogs in which obstruction was produced under procaine did not show

any great difference from those operated on under ether and, indeed, in the instance already mentioned in which the intra-intestinal pressure was higher than usual during the first twenty-four hours and reached 36 cm. of water in six hours, ether had been used. Over this early period, there was no regularity of peristaltic action, as was found subsequently.

During the second twenty-four hours of obstruction, the sustained pressure remained between 6 and 8 cm. of water, while the degree of

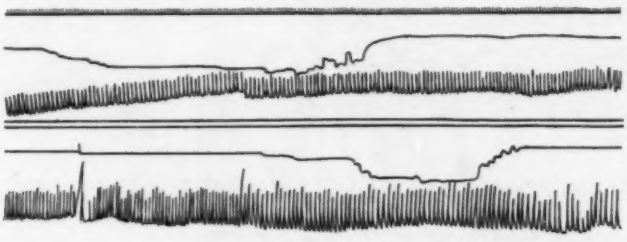

Fig. 2.—Tracing to illustrate the independence of the intra-intestinal pressure as compared with the respirations. Record of animal O. P. 15, in which obstruction had been produced in the lower part of the ileum for ninety-six hours; timer 3 seconds. A rise in the intra-intestinal pressure is represented on all the tracings by descent of the graph toward the bottom of the record.

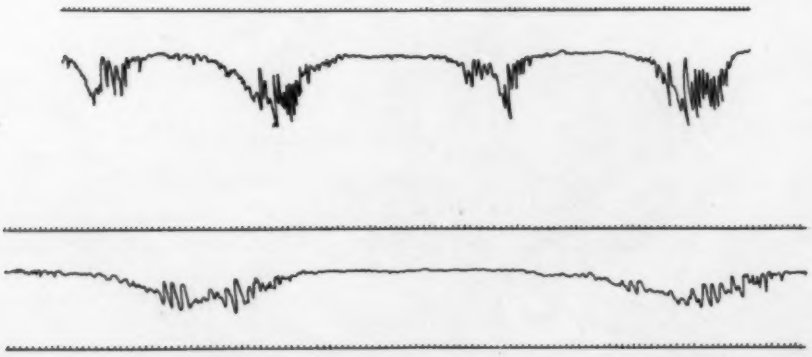

Fig. 3.—Graphic record obtained from one animal with a high intestinal obstruction and one with a low obstruction. The upper tracing is from animal O. P. 26, in which obstruction had been produced in the upper part of the jejunum for fifty-seven hours; the lower tracing is from animal O. P. 30, in which obstruction had been produced in the lower part of the ileum for fifty-two hours; timer 6 seconds.

pressure reached at the maximum of peristalsis rose considerably. In the dogs in which the high obstructions were produced, this pressure varied from 30 to 45 cm., while it generally took until the third day for the pressure in the dogs with low obstruction to reach the same

level. The regularity of the peristaltic action became striking during this same interval. The periods of increased pressure in animals with high obstruction occurred about every third minute and lasted for about two minutes. In the animals with low obstruction, the regularity of action came on from forty-eight to seventy-two hours after the operation and the periods of increased pressure occurred every five to six minutes and lasted about three minutes. In other words, the periods of peristaltic action are more frequent in animals with high obstruction, and the graphic record obtained differed from that made in animals with

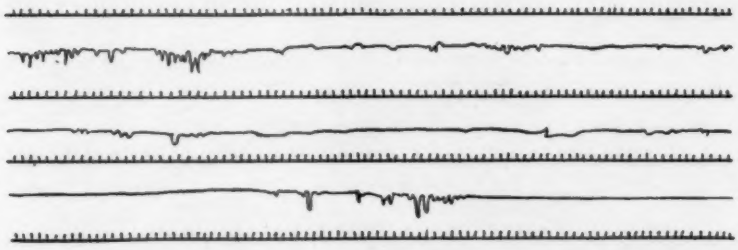

Fig. 4.—Graphic record from animal O. P. 26, after obstruction had been produced in the upper part of jejunum for four hours; timer 6 seconds.

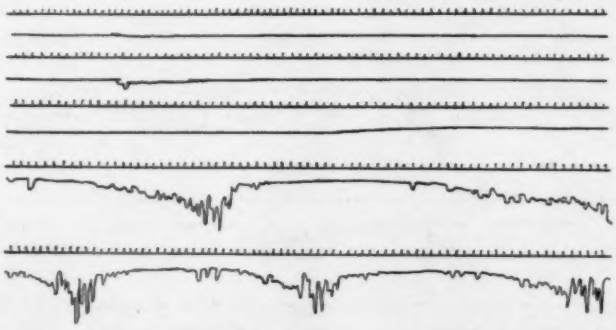

Fig. 5.—The upper three tracings show results in animal O. P. 26, after obstruction had been produced for ten hours; the lower two tracings, after twenty-two hours of obstruction; timer 6 seconds.

low obstruction in that a shorter time was taken for the pressure to reach its maximum. This is best shown in the kymographic records (fig. 3). The lower tracing, from an animal with low obstruction, shows a gradual increase in pressure with little irregularity until after the maximum is reached. The upper record, on the other hand, shows an abrupt or moderately abrupt rise in pressure with considerable irregularity. The fall in pressure in each case is much the same, showing marked evidence of peristaltic action, though somewhat more prolonged in the record made on animals with low obstruction.

On the fourth day, the periods of increased pressure in the animals with high obstruction became less and less numerous, though the maximum level reached from 45 to 50 cm. of water. The sustained pressure began to decline, the toxicity of the animal increased quickly and death usually ensued about ninety-six hours after the obstruction. The dogs with low obstruction lived until the sixth or seventh day. The bowel showed good tone and considerable activity during the fourth and fifth days, after which fewer periods of increased pressure were noted and

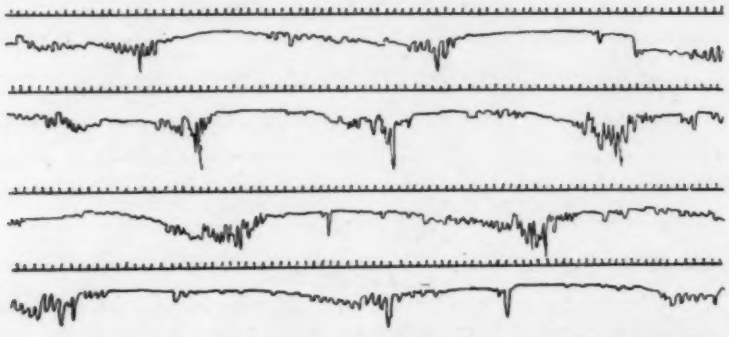

Fig. 6.—Record of animal O. P. 26 after obstruction had been produced for twenty-four hours; timer 6 seconds.

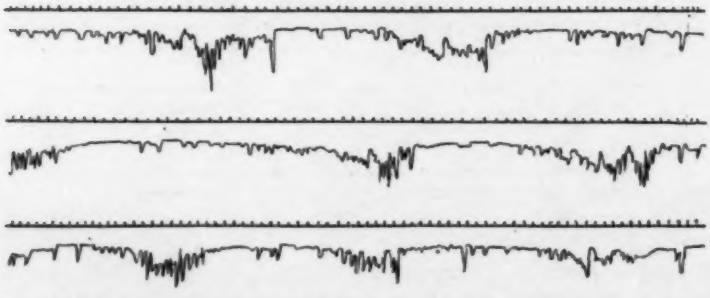

Fig. 7.—Record of animal O. P. 26 after obstruction had been produced for twenty-eight hours; timer 6 seconds.

the maximum level became less. The sustained pressure between these periods also diminished as the toxicity increased. During the twenty-four hours preceding death, the graphs showed slight undulations as the only evidence of intestinal motility.

The degree of pressure, then, within the lumen of the bowel in obstruction is greatly increased over the normal. The maximum levels in animals with high and low obstruction do not show great differences. The pressure rarely rises above 50 cm. of water, though in one isolated

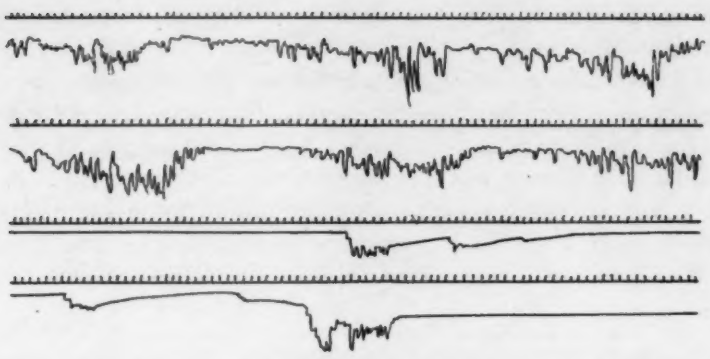

Fig. 8.—The upper two tracings show record of animal O. P. 26 after obstruction had been produced for thirty-three hours; the lower two tracings, after forty-eight hours; timer 6 seconds.

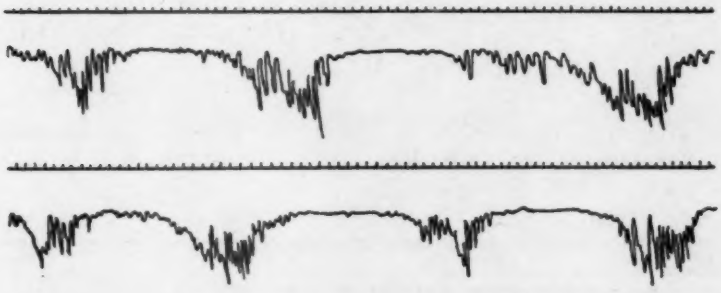

Fig. 9.—Record of animal O. P. 26 after obstruction had been produced for fifty-seven hours; timer 6 seconds.

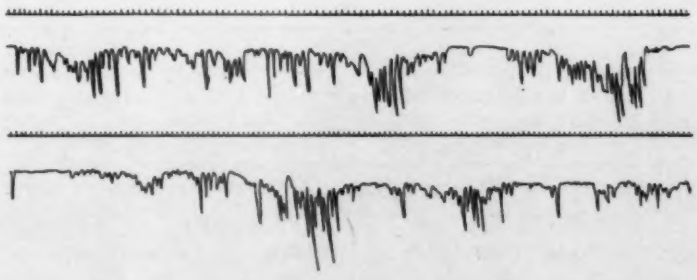

Fig. 10.—Record of animal O. P. 26 after obstruction had been produced for seventy-one hours; timer 6 seconds.

instance a pressure of 58 cm. was reached. When accompanied by retching, vomiting or straining, the pressure is greatly increased and will reach above 100 cm.

In two instances, the intra-abdominal pressure was determined along with the intra-intestinal pressure. This was done by means of small soft rubber bags that were filled with water and placed within the abdomen; a connection was then made to a water manometer. A slight positive pressure was present when the animal was lying down; it measured from 1 to 4 cm. On deep inspiration, this level reached 8 cm. These variations were independent of the intra-intestinal pressure.

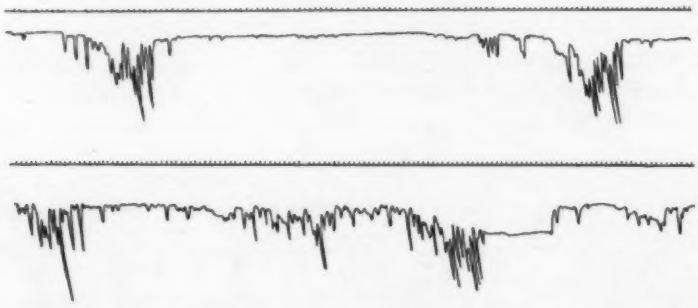

Fig. 11.—Record of animal O. P. 26 after obstruction had been produced for seventy-five hours; timer 6 seconds.

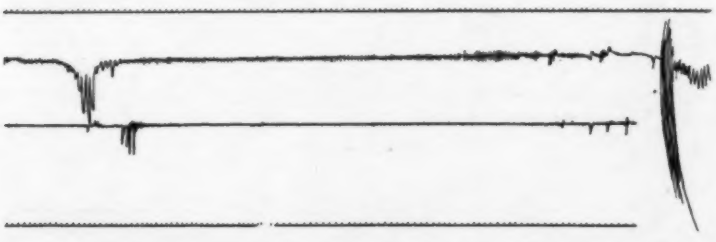

Fig. 12.—Record of animal O. P. 19 in which obstruction had been produced in the upper part of the jejunum for one hundred and thirty hours. Animal was moribund. Results show effect of respirations and vomiting; timer 6 seconds.

That the changes in the latter pressure are independent of the respirations is shown in figure 2. Extreme changes in intra-abdominal pressure, resulting from retching, vomiting and other conditions, however, are readily transmitted to within the lumen of the bowel (fig. 12).

In four instances, the pressure was measured within the lumen of isolated portions of the intestine. In three cases, these loops were made up of portions of duodenum and jejunum and in one instance of jejunum alone. Within the first twelve hours in all cases, the pressure

had risen to at least 30 cm. of water, and by twenty-four hours it had reached about 50 cm. The rhythmic periods of increased pressure were likewise present, and the sustained pressure between them showed a high tension, measuring from 16 to 18 cm. The sustained pressure

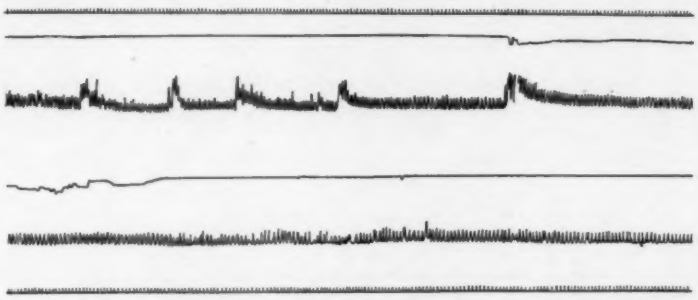

Fig. 13.—Graphic record from animal O. P. 17 in which obstruction had been produced in the lower part of the ileum for twenty-four hours. Respirations were recorded; timer 3 seconds.

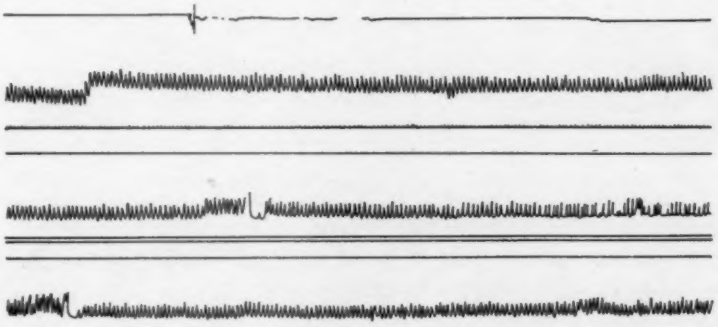

Fig. 14.—Record from animal O. P. 17 after obstruction had been produced for forty-eight hours. Respirations were recorded; timer 3 seconds.

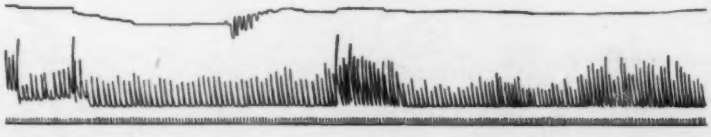

Fig. 15.—Record from animal O. P. 17 after obstruction had been produced for seventy-six hours. Respirations were recorded; timer 3 seconds.

was maintained at a higher level than in the animal with simple obstructions and did not fall off until perforation occurred. The maximum pressure observed was 70 cm. of water. The length of life in these animals was from twenty-nine to forty-eight hours. Three died from general peritonitis resulting from a perforation of the loop at the

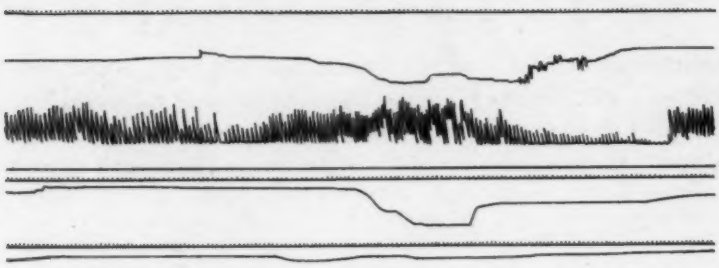

Fig. 16.—The upper tracing shows record from animal O. P. 17 after obstruction had been produced for ninety-six hours; the lower tracing, after one hundred hours; timer 3 seconds.

Fig. 17.—Record from animal O. P. 17 after obstruction had been produced for one hundred and twenty-four hours; timer 3 seconds.

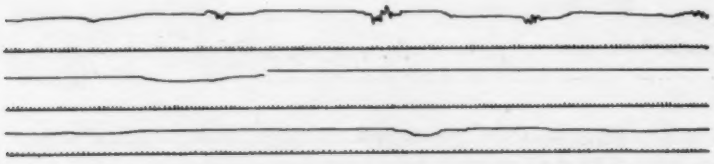

Fig. 18.—Record from animal O. P. 17 after obstruction had been produced for one hundred and fifty hours; timer 6 seconds.

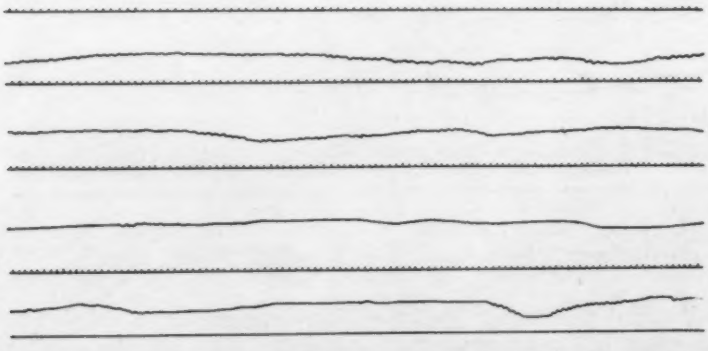

Fig. 19.—The upper two tracings show record from animal O. P. 17 after obstruction had been produced for two hundred and sixteen hours; the lower two tracings, after two hundred and twenty-one hours. The results show effect of fecal fistula; timer 6 seconds.

antimesenteric border. In the fourth there was a leak about the apparatus and peritonitis resulted.

In the animals with simple obstruction, perforation or gangrene of the antimesenteric border was not encountered, as in the experiment on animals in which a loop was made. This is of interest, for in the

TABLE 1.—*Data for Dogs Used in Experiment*

No.	Serial No.	Anesthetic	Site of Obstruction	Duration of Life, Hours	Cause of Death
1	O.P. 6	Morphine and ether	Upper part of jejunum	56+	Toxemia and general peritonitis
2	O.P. 8	Morphine and ether	Lower part of ileum	73+	Toxemia and local peritonitis
3	O.P. 9	Morphine and ether	Middle small bowel	102+	Toxemia and distemper
4	O.P. 10	Procaine	Upper part of jejunum	100+	Toxemia and local peritonitis
5	O.P. 11	Procaine	Middle small bowel	53+	Toxemia and local peritonitis
6	O.P. 12	Procaine	Lower part of ileum	77+	General peritonitis and distemper
7	O.P. 13	Morphine and ether	Middle small bowel	103+	Toxemia and local peritonitis
8	O.P. 14	Morphine and ether	Upper part of jejunum	70+	Toxemia and local peritonitis
9	O.P. 15	Morphine and ether	Lower part of ileum	121	Toxemia and general peritonitis
10	O.P. 16	Morphine and ether	Lower part of ileum	75+	Toxemia and general peritonitis
11	O.P. 17	Morphine and ether	Lower part of ileum	221+	Toxemia and general peritonitis
12	O.P. 18	Morphine and ether	Middle small bowel	60	Toxemia and general peritonitis
13	O.P. 19	Morphine and ether	Upper part of jejunum	130+	Toxemia and local peritonitis
14	O.P. 20	Morphine and ether	Isolated loop in duodenojejunum	29	General peritonitis
15	O.P. 21	Morphine and ether	Isolated loop in jejunum	48	General peritonitis
16	O.P. 22	Morphine and ether	Upper part of jejunum	56	Toxemia and general peritonitis
17	O.P. 23	Morphine and ether	Isolated loop in duodenojejunum	54+	General peritonitis
18	O.P. 24	Morphine and ether	Isolated loop in duodenojejunum	45	General peritonitis
19	O.P. 25	Procaine	Lower part of ileum	29	General peritonitis (chloroformed)
20	O.P. 26	Procaine	Upper part of jejunum	76+	Toxemia
21	O.P. 27	Procaine	Lower part of ileum	140+	Toxemia
22	O.P. 30	Morphine and ether	Lower part of ileum	216	Chloroformed (fecal fistula)

latter instance we find that the pressure rose more quickly and reached a higher level, while the sustained pressure between the periods of increased tension was more than twice that found in the animals in which simple obstruction was produced.

Most of the animals, as is shown in table 1, died of a complicating peritonitis. There can be little doubt that this would materially affect the intestinal motility and probably cause the fall in pressure that is

TABLE 2.—*Protocol of Dog O. P. 17*

Period of Observation	Hours Obstructed	Maximum-Minimum Pressure, Cm. Water: Maximum	Maximum-Minimum Pressure, Cm. Water: Minimum	Notes
Feb. 10—11:00-12:00 a. m.	...	..	..	Operation; ¼ grain morphine; ether; obstruction lower part of ileum
4:00- 4:15 p. m.	4	14	10	
Feb. 11—11:00-12:00 a. m.	24	18	7	Fig. 13
10:00-11:00 p. m.	35	18	7	
Feb. 12—12:00- 1:00 p. m.	49	9	5	Fig. 14
4:00- 5:00 p. m.	52	12	8	
Feb. 13—11 a. m.-12:30 p. m.	72	36+	7	
3:00- 4:00 p. m.	76	44	5	Fig. 15
Feb. 14—10:30-11:15 a. m.	96	40	8	Fig. 16
3:00- 4:00 p. m.	100	28	8	Fig. 16
Feb. 15—12:00- 1:00 p. m.	121	20	6	
2:30- 4:00 p. m.	124	30	6	Fig. 17
8:30- 9:30 p. m.	130	24	8	
Feb. 16—10:00-11:30 a. m.	144	16	6	
3:45- 5:15 p. m.	150	24	10	Fig. 18
8:30- 9:30 p. m.	154	18	10	
Feb. 17—11:00-12:00 a. m.	168	18	8	Fecal fistula
4:30- 6:00 p. m.	174	30	2	
8:45-10:00 p. m.	177	16	8	
Feb. 18— 2:50- 4:00 p. m.	196	18	2	
Feb. 19—11:00-12:00 a. m.	216	14	10	Fig. 19
5:00- 6:00 p. m.	221	24	12	Fig. 19
Feb. 20— 4:00- 5:00 p. m.	244	No record		Fig. 20
Feb. 21— 7:00 a. m.	...	..	..	Found dead; autopsy: general peritonitis

TABLE 3.—*Protocol of Dog O. P. 20*

Period of Observation	Hours Obstructed	Maximum-Minimum Pressure, Cm. Water: Maximum	Maximum-Minimum Pressure, Cm. Water: Minimum	Notes
Feb. 16—11:00-12:00 a. m.	..	..	..	Operation; ¼ grain morphine; ether; isolated loop in duodenojejunum; no anastomosis
11:00-12:00 p. m.	12	30	12	Fig. 21
Feb. 17—10:00-11:30 a. m.	24	36	13	Figs. 21 and 22
2:00- 3:00 p. m.	27	44	14	Fig. 22
5:00 p. m.	29	..	..	Dead; general peritonitis; perforation of loop due to apparatus

TABLE 4.—*Protocol of Dog O. P. 26*

Period of Observation	Hours Obstructed	Maximum-Minimum Pressure, Cm. Water: Maximum	Maximum-Minimum Pressure, Cm. Water: Minimum	Notes
Feb. 27—11:00-12:00 a. m.	..	..	..	Operation; procaine 2%; obstruction in upper part of jejunum
4:00- 5:30 p. m.	4	17	5	5 cc. cold water injected into bowel; fig. 4
9:30-10:30 p. m.	10	8	5	Fig. 5
Feb. 28—10:30 a. m.-1 p. m.	24	36	5	Figs. 5 and 6
3:45- 4:45 p. m.	28	30	6	Fig. 7
9:00-10:00 p. m.	33	30	6	Fig. 8
Feb. 29—12:00- 1:00 p. m.	48	28	10	Fig. 8
3:00- 4:30 p. m.	51	30	6	
8:30- 9:30 p. m.	57	54	6	Fig. 9
March 1—10:00-11:00 a. m.	71	54	6	Fig. 10
3:00- 4:00 p. m.	75	58	6	Fig. 11
March 2— 7:00 a. m.	..	..	..	Found dead; autopsy showed local adhesions only

so marked during the final twenty-four hours. That this is the case might be concluded from the graphs obtained from the animals with high obstruction that are shown in figures from 4 to 11. The last graph was obtained within sixteen hours of death, and the intestinal motility, as is shown by the records, was nearly as active as at any earlier period.

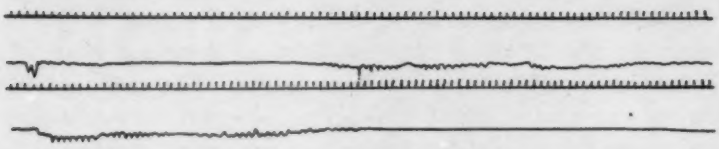

Fig. 20.—Record from animal O. P. 17 after obstruction had been produced for two hundred and forty-four hours; timer 6 seconds.

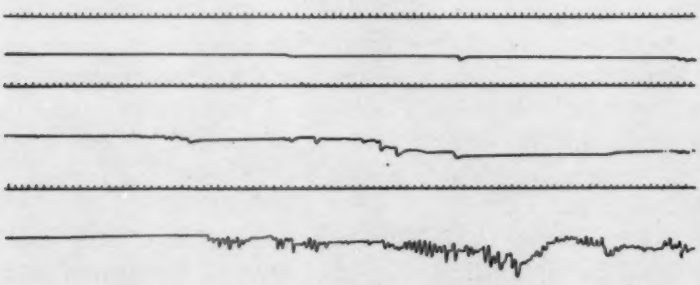

Fig. 21.—The upper two tracings show record from animal O. P. 20 after isolated loop had been produced in the duodenojejunum for twelve hours; the lower tracing, after twenty-three hours; timer 6 seconds.

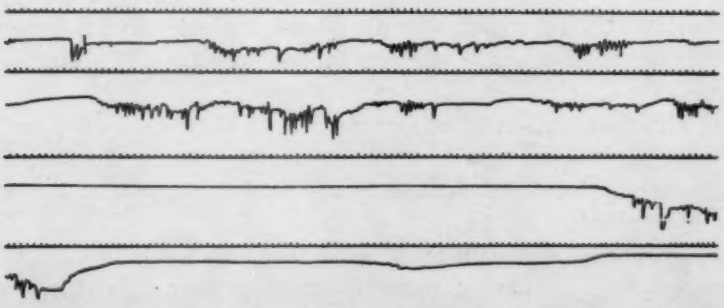

Fig. 22.—The upper two tracings show record from animal O. P. 20 after isolated loop had been produced for twenty-four hours; the lower two tracings, after twenty-seven hours; timer 6 seconds.

Autopsy did not reveal general peritonitis and no local adhesions or exudation beyond what one might expect after such manipulation. Death was thought to be due, therefore, in most part to the toxemia of obstruction. Figure 12 was obtained from another animal with high obstruction that, as shown at autopsy, had general peritonitis. The

graph was taken within two or three hours of death while the animal was moribund, but it shows what we found the more common record of the intestinal motor activity before death. Without a complicating peritonitis, then, we feel all our graphs would show more evidence of peristalsis than they have done within twenty-four hours of death, but that even without peritonitis the pressure falls and the motility becomes less with the increasing toxemia of obstruction.

Figures 19 and 20 are of interest as they show the changes that come from the development of a fecal fistula. The drainage in this case was not sufficient to empty the obstructed bowel, so that a pressure was kept above the normal but below that within an obstructed bowel without a fistula. A well marked change in the character of the graphs is noted.

SUMMARY

The conclusions that follow from these experiments seem to show that in small dogs with simple obstruction the intra-intestinal pressure is maintained at a level (from 6 to 8 cm. of water) about twice that of the normal maximum (from 2 to 4 cm. of water). While the bowel is active, the pressure rises to ten or fifteen times the normal (from 30 to 60 cm. of water).

What we consider a fairly exact conception of the type and magnitude of the intestinal motility in obstruction throughout its course is shown in the accompanying tracings. Three periods may be roughly distinguished. The first, which embraces the twenty-four hours following obstruction, shows little change from the normal. The second period shows a rise in the basic or sustained pressure with marked peristalsis and a great increase in the intra-intestinal pressure generally. The third or terminal phase shows a falling pressure and a decrease in the peristaltic activity.

The fact that periods of violent peristalsis come on earlier and are more frequent in animals with high obstruction than in those with low obstruction may have some clinical value in helping one to locate the site of obstruction.

In experiments in which an isolated loop was made, the degree of pressure reached (70 cm. of water) is much greater than that in dogs with simple intestinal obstruction.

THIRTY-SIXTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

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CONGENITAL DEFORMITIES

Premature Ossification of Lower Epiphysis of the Tibia Following Forcible Correction of Clubfeet.—Riedel [1] observed five patients who showed premature ossification of the lower tibial epiphyses following forcible manipulation for the correction of congenital talipes equinovarus. Each patient came under observation because of a persistent tendency toward recurrence of the deformity. In all cases, roentgen-ray examination showed that the lower tibial epiphyses had closed, while the lower epiphyses of the fibula were still open. The tibiae had stopped growing, while the fibulae were still elongating, and the asymmetrical growth resulted in tilting the astragalus and supinating the os calcis and the whole tarsus. In all of the cases the original manipulation had been forcible, the wrench having been used. Presumably the epyphyses had been injured during the procedure. Riedel believed that many cases of recurring clubfoot are to be explained in this manner. He stated that he has seen total necrosis of the astragalus result from a forcible correction of a clubfoot.

DISTURBANCES OF GROWTH

Genu Varum and Genu Valgum Infantum.—Boehm [2] has been convinced that many cases of genu varum and valgum are not due to rickets, and has made an investigation to find other possible causes of the deformities. His studies led him to the conclusion that the non-

* This Report of Progress is based on a review of 258 articles selected from 419 titles dealing with orthopedic surgery appearing in the medical literature between Sept. 24, 1927, and Feb. 25, 1928. Only those papers that seem to represent progress have been selected for note and comment.

1. Riedel: Zentralbl. f. Chir. **55**:951, 1928.
2. Boehm, M.: Ztschr. f. orthop. Chir. **49**:321, 1928.

rachitic genu varum and valgum represent a regressive phase or variation in the development of growth of the skeleton of the lower extremities. In the new-born child, the knee is in a position of flexion, outward rotation and lateral declination in relation to the general alinement of the leg. Boehm has shown from studies of many children and of anatomic specimens that during the first five years of growth the alinement of the leg changes, the knee rotating slightly inward, losing its flexion and assuming a slight medial declination. However, variations may occur and in certain persons these may be of a regressive type, leading eventually to the formation of genu valgum or varum. Static and mechanical forces resulting from weight bearing were regarded by Boehm as important, not as primary factors in influencing the development of the deformities but as occurring secondarily after the variation in the anatomic development of the leg has been produced.

RICKETS

Historical.—Writing of the history of the knowledge of rickets, Foote [3] stated that when Whistler published his thesis in 1645, and Glisson his book in 1650, it was called an entirely new disease. According to present theories of cause, there is no reason to suppose that the disease did not exist before 1650. Swaddling was a method employed by ancient peoples to prevent the development of deformities. Soranus of Epiphysus apparently wrote about the disease in the second century A. D. The author has observed in the Army Medical Museum at Washington the skull of a pre-Columbian Peruvian boy with typical rachitic changes. Between 1450 and 1500, Bruges described the disease in England. Portraits by Lochner, Strigal, Bergmaier and others show rachitic changes. Foote concluded that the disease undoubtedly existed at all times and periods.

Sunshine and Skyshine.—Tisdall and Brown [4] made a study of the effect of seasonal variation on the antirachitic value of sunshine in the latitude of Toronto. Experiments were conducted on albino rats which were kept on a constant rachitogenic diet. The rats were exposed two hours daily and the results appraised from study of roentgenograms and analysis of the calcium and phosphorus in the blood and bone. The authors concluded that the sun's rays in the vicinity of Toronto have slight but definite antirachitic effect, and that there is a sharp increase in the antirachitic effect of sunshine, beginning in March. In April and May, the antirachitic effect is approximately eight times as great as in

3. Foote, J. A.: Evidence of Rickets Prior to 1650, Am. J. Dis. Child. **34**: 443 (Sept.) 1927.

4. Tisdall, F. F., and Brown, A.: Seasonal Variation of Antirachitic Effect of Sunshine, Am. J. Dis. Child. **34**:721 (Nov.) 1927.

December, January and February. These results agree with those of Dorno, who found the ultraviolet content of midday sun in the Alps in January only 10 per cent as great as in July.

Similar methods were employed by the same authors [4a] in testing out the antirachitic action of skyshine. The rats were exposed on the shady side of the roof. The authors concluded that the effect of skyshine is from about one-half to two-thirds as great as that produced by sunshine.

Hess and Anderson,[5] by an improved technic, showed that ultraviolet radiation with rays shorter in length than those of sunshine is more potent in healing rickets than the effective area of solar rays. The effective band comprises the waves between 290 and 313 millimicrons. The studies brought out the fact that the area of specific solar radiation is markedly circumscribed, and that a difference of a few millimicrons or millionths of a millimeter determines whether or not waves are effective. During the winter months when the shorter ultraviolet rays do not reach the earth and the longer ones are less intense, the width of the antirachitic zone of sunlight is only about 5 millimicrons. These observations emphasize the danger of the small band of specific radiation being filtered from the atmosphere by moisture, dust, smoke, and other foreign substances.

Tisdall and Brown [6] tested the antirachitic value of the sun's rays when passed through various special window glasses. These included Vitaglass, Vicray glass and Corning glass. They concluded that the effect obtained was from 25 to 50 per cent of that obtained without the use of glass. An obvious difference could not be detected between the three glasses. The antirachitic effect of sunshine which had passed through ordinary glass was shown to be negligible except immediately adjacent to the window. The authors stated that with any type of glass it is necessary to employ the direct rays of the sun in order to receive much benefit, and that in winter the use of special glass is probably of little benefit.

Robertson [7] reported an investigation of the effect of exposure to the sun on susceptibility of rachitic rats to infection. The organism used as the infecting agent in the experiments was a gram-negative bacillus isolated from the nose of a normal rat. The author showed

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4a. Tisdall, F. F., and Brown, A.: Antirachitic Effect of Skyshine, Am. J. Dis. Child. **34**:737 (Nov.) 1927.

5. Hess, Alfred F., and Anderson, William, T.: Antirachitic Activity of Monochromatic and Regional Ultraviolet Radiations, J. A. M. A. **89**:1222 (Oct. 8) 1927.

6. Tisdall, F. F., and Brown, A.: Antirachitic Effect of the Sun's Rays Through Various Special Glasses, Am. J. Dis. Child. **34**:742 (Nov.) 1927.

7. Robertson, E. C.: Canad. M. A. J. **17**:1033 (Sept.) 1927.

that twice as many rats which had been exposed to the sun survived intraperitoneal infection as those which had not been exposed.

[Ed. Note.—The articles summarized reporting the results of investigations of the antirachitic properties of radiation are only a few of a great many dealing with different phases of the same general subject that have been published in the last few years. Little by little the knowledge of the action and effects of radiation is being increased, and already a certain amount of order has emerged from what a short time ago was chaos. While the subject is being approached chiefly from its relation to rickets, the orthopedic surgeon cannot lose sight of the larger bearing of radiation in relation to the function of bone and the treatment of bone diseases in general. It is reasonable to suppose that increase of knowledge concerning radiation in rickets may also have a bearing on the employment of light therapy in other conditions.]

Rachitic Deformities of the Lower Extremities.—Moore [8] made a study of rickets in the lower extremities. He found that in normal legs the approximating epiphyseal plates of the femur and tibia are parallel and that when the inner borders of the feet are parallel both the knees and the ankles are in contact. In a typical example of rickets, he found that the epiphyseal lines of each knee, if extended, would meet at an acute angle. With the planes of the lower femoral and upper tibial epiphyses coming together on the outside, knockknee would naturally result. The vacant space between the epiphyses in the open part of the angle gradually fills up with calcified bone and new epiphyseal planes are formed, parallel to each other but at an abnormal angle to the long axis of the diaphyses of both the tibia and the femur. The author stated that until such a joint becomes fixed in a normal or a valgum or varum position a decided lateral mobility is demonstrable. This exaggerated mobility of the knee is often the first sign of rickets in the leg. A normal knee gives a lateral movement of 3 cm. or less as measured at the heel, regardless of the length of the leg. A knee mobility arthrometer was devised so that mathematically accurate measurements could be obtained each month and become an index of the amount of lateral mobility of the knee. According to the author, this index may be used to determine whether or not genu valgum or varum is imminent. After such a deformity has developed, its value lies in determining whether the joint has become too set to be capable of correction by brace treatment.

DISTURBANCES OF DEVELOPMENT AND GROWTH

The Ovary in Osteomalacia.—Writing on the relation of the ovary to osteomalacia, Fraser [9] stated that a close relationship between the ovary, pregnancy and osteomalacia has been recognized since Fehling

8. Moore, Ulysses C.: J. Bone & Joint Surg. **10**:96 (Jan.) 1928, no. 1.
9. Fraser, J. R.: Am. J. Obst. & Gynec. **14**:697 (Dec.) 1927.

introduced castration in 1887. Experimental work has shown that the ovaries exert a marked influence on phosphorus metabolism. Blair Bell mentioned a 50 per cent reduction in excretion of calcium after ovariotomy. Fraser described the changes noted in an ovary removed in midpregnancy from a patient with well marked osteomalacia. Not only the cortex but the entire ovary showed a marked hyperplasia of the glandular and interglandular tissue. There was a general venous congestion and widespread formation of follicles. All stages of maturation of the follicles were seen. Many atritic follicles were present, many of them cystic and with proliferation of the thecal cells. There were more large vessels present than is usually found, particularly in the medulla. This may merely represent a further stage of what normally occurs during pregnancy. The author expressed the belief that the ovary plays a part in osteomalacia because of the therapeutic benefit following castration, the frequent occurrence and aggravation of the disease with pregnancy, the high degree of fertility in this disease, and the changes which the author has described.

Osteogenesis Imperfecta.—Welz and Lieberman [10] reported the occurrence of osteogenesis imperfecta in single ovum twins. The first infant died in nine hours and the second in fourteen hours. The blood calcium in the mother was 8.9 mg. per hundred cubic centimeters of blood, and in the second infant was 10.1 mg. per hundred cubic centimeters of blood. The Wassermann reaction was negative. Roentgen-ray examination of the second infant showed fractures in nearly every bone in both the upper and the lower extremities, in various stages of healing, also healed fractures of most of the ribs on both sides. Marked deformities were present in both femora. There was no evidence of injury to the spine. The clavicles were not broken. The epiphyses of the tibia showed a dense straight line of bone deposit. Roentgen-ray examination of the first infant showed an almost identical appearance. In this case, however, the clavicles were broken.

Brittle Bones and Blue Sclera.—Colby [11] reported that there had been brought to his attention three members in one family with brittle bones and blue sclerae. One child had blue sclerae but had not had any fractures. Another child, a girl, aged 5 years, had had four previous fractures from slight trauma. Discussing the disease in general, the author said that it is usually called osteogenesis imperfecta and that it is characterized by brittle bones, blue sclerae, deafness and hypermobility of the joints. It was first described by Von Ammon in 1839. It is transmitted by direct inheritance, and children of affected parents who

10. Welz, W. E., and Lieberman, B. L.: Am. J. Obst. & Gynec. **14**:49 (July) 1927.

11. Colby, E. G.: Southwestern Med. **11**:36 (Aug.) 1927.

do not have blue sclerae do not transmit the disease to their offspring. Little is known concerning treatment, and all present methods are of practically no avail.

Bone Tissue in Conditions of Circulatory Disturbance.—Mueller [12] studied bone tissue from persons with disturbed circulation, such as occurs in arteriosclerosis, and found definite structural changes. The intermediary lamellae showed areas of necrosis, while the lamellar systems surrounding central vessels and marrow spaces were essentially normal. The necrotic areas were found only in the compact bone and were totally lacking in the spongy bone. In senile bone without accompanying arteriosclerosis necrotic areas were not seen. Bone proliferation and evidence of reaction such as are usually seen in the vicinity of necrotic bone areas were not seen in the specimens of bone from patients with circulatory disturbance.

SCOLIOSIS

Lucas [13] stated that while he considered the treatment of structural scoliosis still far from satisfactory, he had employed Steindler's method with good results. By this method the proper body alinement is maintained by the formation of compensatory curves above and below the primary curve. Direct force is not applied to the primary curve. The first stage of the treatment lasts from one to two months and consists of gymnastics for stretching and loosening the spine and of extension on a Bradford frame. Extension is obtained by applying about 10 pounds to the head and from 30 to 40 pounds to the pelvis. In case there is much deformity of the chest, blowing on Wolff's bottles is prescribed. The second stage is one of fixation and lasts from two to three months. A series of plaster jackets is applied, the child being suspended with the head sling. The arm on the concave side is abducted and the hip on this side adducted. If at the end of this time the body alinement is good, the spine stable with good compensatory curves and the muscles sufficiently strong, the child is sent home in a brace. If the spine is unstable, a fusion operation is performed by Hibbs' method. After operation, the child is kept recumbent for three months with plaster fixation; then a brace is worn for one year longer.

Summarizing an article on scoliosis, Ober and Ghormley [14] stated that functional and mild structural curves can be managed by simple corrective exercises and apparatus. Operative measures are to be recommended in adult patients whose spines are not already fixed by

12. Mueller, W.: Beitr. z. klin. Chir. **138**:494, 1926.

13. Lucas, L. S.: Northwest Med. **26**:588 (Dec.) 1927.

14. Ober, Frank R., and Ghormley, Ralph K.: Scoliosis, J. A. M. A. **90**:361 (Feb. 4) 1928.

nature and who are doomed to wear a jacket for life. Fusion operations should not be performed on growing children except in paralytic conditions and then only in case of severe deformity which cannot be held by other measures. In the authors' opinion, the principal indications for operative treatment are: (1) rapid increase of the curve; (2) signs of general debility or ill health; (3) rib pressure with intercostal or pelvic pain, and (4) inability to hold the curve in persons with infantile paralysis. The first three indications are for adults; the fourth is for children or adults.

[ED. NOTE.—We are in agreement with the opinions of the last authors but would supplement their operative indications by adding cases in which the curve is the result of an anatomic defect such as a wedge-shaped dorsal vertebra. Experience indicates that such curves increase with the growth of the patient irrespective of all corrective measures except fusion operations, and sometimes even in spite of them.]

PAIN IN THE BACK

Abdominal Symptoms Associated with Chronic Strain of the Spine.—Carnett [15] expressed the belief that midline abdominal pain may be caused by chronic strain of the lumbar spine and sacro-iliac joints. He observed the condition repeatedly. It is associated with an increased lumbar lordosis and increased dorsal kyphosis. Midline tenderness is found sharply limited to the area of the intra-abdominal portion of the vertebral bodies, and all aspects of the vertebrae and intervertebral disks that are accessible to palpation are uniformly tender. The majority of the patients who exhibit midline tenderness also have an isolated area of tenderness in each iliac fossa over the superior portion of the sacro-iliac joint. The midline tenderness has often been considered aortic tenderness and also sometimes mistaken for tenderness due to appendicitis. In many of the patients with exaggerated lumbar lordosis, the dominant symptoms are those of irritation of the intercostal nerves, which is more or less widespread but is manifested mainly by pain and tenderness over the abdomen, particularly in the right lower quadrant.

Pain in the Lower Part of the Back in Connection with Pelvic Disorders.—The various causes of backache have been considered by Kreuscher,[16] and he has pointed out the infrequency of the production of this symptom by gynecologic disorders. His chief argument against any relationship between uterine conditions and pain in the back is that the innervation of the uterus is derived from the pelvic plexus, the ovarian plexus and a few filaments from the sacral nerves. Consequently,

15. Carnett, John Berton: Ann. Surg. **85**:509 (April) 1927.
16. Kreuscher, P. H.: Surg. Gynec. Obst. **45**:482 (Oct.) 1927.

there is no logical reason for the transmission of pelvic pain into the lower lumbar and sacro-iliac regions.

Etiologic Factors in Sciatic Scoliosis.—That fecal retention in the large bowel may be an etiologic factor in the production of certain disease symptoms, particularly of the syndrome of the so-called sciatic scoliosis, has impressed Osgood [17] from his experience in treating some of these patients. He has made a detailed report of six patients showing this symptom-complex. The duration of the symptoms was very long. All of the usual methods of orthopedic treatment had been tried without avail. Relief was finally obtained from measures directed toward the correction of colonic stasis and fecal retention, such as intestinal massage and colonic irrigations.

Abnormalities of the Lumbar Spine.—O'Reilly [18] continued his investigations of the anatomic abnormalities of the lower spine. He found them present in a high percentage of cases. In a series of 100 roentgenograms, some abnormality was present in 33 per cent, and in an examination of dried bones, abnormalities were found in 17 per cent. The most common abnormality was sacralization of the transverse process of the fifth lumbar vertebra, either fused to the sacrum or articulating with it. The author pointed out the fact that the presence of abnormalities in the so-called normal back frequently leads to legal complications after an injury. In order to prevent this and to protect the workman with a potentially weak back from serious strain, O'Reilly proposed that all workers whose occupations might result in back strain should have a roentgenogram of the spine made before being employed.

Operation for Extra-Articular Fusion of the Sacro-Iliac Joint.—An operation for extra-articular fusion of the sacro-iliac joint was reported by Campbell.[19] The essential feature is the transplantation of a portion of the posterior crest of the ilium into the posterior sacro-iliac fossa and packing it about with bone chips. The postoperative treatment consists of recumbency on a Bradford frame for a period of six weeks. Thus far the author has performed the operation on seven patients. Of these, five are apparently well, and in the other two the operation has been performed too recently to permit an estimation of the result.

[ED. NOTE.—This operation appears somewhat similar to one originated by Dr. E. G. Brackett about fifteen years ago. He used a transplant from the tibia, while Campbell employed a bone graft from the crest of the ilium. We believe that Brackett has since abandoned his operation in favor of Smith-Petersen's operation.]

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17. Osgood, Robert B.: J. Bone & Joint Surg. **9**:667 (Oct.) 1927.

18. O'Reilly, Archer: Abnormalities of Lower Part of Back, J. A. M. A. **89**: 1128 (Oct. 1) 1927.

19. Campbell, W. C.: Surg. Gynec. Obst. **45**:218 (Aug.) 1927.

MECHANICAL DISTURBANCES, ANATOMIC INVESTIGATIONS, CERVICAL RIBS

Cervical Ribs.—From a study of patients with cervical ribs treated at the Mayo Clinic, Adson and Coffee [20] drew the following conclusions: In about 55 per cent of the cases cervical ribs do not cause any symptoms, and patients in whom the condition is discovered purely by accident ought not to be informed of it, as this knowledge is apt to give rise to neuroses with symptoms referable to the rib. The surgical indications depend on the degree and the character of the incapacity produced by the presence of pain, hyperesthesia, anesthesia and circulatory disturbance. Surgical treatment ought not to be advised for the relief of mild, indefinite pain in the neck and shoulder. The authors consider that the anterior approach and tenotomy of the scalenus anticus muscle are preferable to the transcervical approach and resection of the cervical rib, since the same relief is obtained, the procedure is less formidable and postoperative numbness in the arm and palsy of the brachial plexus are avoided.

[ED. NOTE.—It is to be remembered that symptoms apparently due to cervical rib may often be relieved by orthopedic treatment directed toward improving posture and relieving irritation and mechanical disturbances of the cervical spine. Such treatment ought to be given a trial before operation is resorted to. As far as not telling the patient of the accidental discovery of a symptomless cervical rib is concerned, we do not agree with the authors. Leaving aside the ethical question which is here raised, we believe there are few patients injured by the frank discussion of the condition found on examination, provided that the harmlessness of the condition is explained.]

Pressure on the Brachial Plexus by the Normal First Rib.—Brickner [21] made a study of patients with symptoms of pressure on the brachial plexus caused by a normal first rib. He reported six cases of his own. In the less severe cases the symptoms are pain in the arm, paresthesia and other phenomena. These may be relieved by elevating the shoulder and by exercising the trapezius to keep the shoulder elevated. Three of the author's cases were of this type, the symptoms probably being due to the dragging of the brachial plexus over the first thoracic rib. In the more severe cases all the phenomena of pressure by a cervical rib are produced by the pressure of an abnormal or even a normal first thoracic rib. Several of the patients required operative treatment and were relieved by resection of the first rib. When an operation is performed on a patient with a cervical rib, it is

20. Adson, Alfred W., and Coffey, Jay R.: Ann. Surg. **85**:839 (June) 1927.

21. Brickner, Walter M.: Ann. Surg. **85**:858 (June) 1927.

important, the author stated, to observe whether the pressure to be relieved is due to the supernumerary rib or to the normal first rib.

The Articular Mechanism of the Diarthroses.—Walmsley [22] reported the results of his study of the articular mechanism of the diarthroses. He stated that at the majority of the diarthroses two functions are equally to be served: (1) the function of weight transmission, which is accomplished in only one position and to secure which there are the articular mechanisms, and (2) the function of movement, during which the surfaces of the elements are moved on one another and maintained in their relations to one another by the mechanisms of movement, which are the related muscles. Each function has its own mechanism. Walmsley said that there is one aspect of mechanism of the joints that has not always been taken into account in descriptions of function of the joints and, in fact, is by no means universally admitted, namely, "The definite and nonreciprocal shape of the joint surfaces which determines that they are in full congruence only in the positions in which they transmit weight and are incongruent in all other positions. . . . At the hip joint as the limb moves toward extension, and materially assisted by the anterior inclination of the neck of the femur, the capsule is progressively twisted and shortened, and, as it were, guides the head of the bone like a screw into its socket until at the position of full extension (about fifteen degrees beyond the vertical) the capsule is taut, the head of the bone is screwed home, the surfaces are fully congruent, and further movement of a larger on to a smaller surface is impossible."

[ED. NOTE.—The author brings out a point in reference to the mechanism of the hip that is most important in the reduction of fractures of the hip, namely, the screwing home action of the motion of extension. To approximate the fragments, it is necessary not only widely to abduct and internally rotate the hip, but also to extend it.]

ARTHRITIS

General Articles.—Pemberton [23] stated his conclusions regarding chronic arthritis after a clinical and biochemical investigation of 1,200 cases. He considered the division of the cases into proliferative and degenerative types the simplest and best classification. He thought that the rôle of focal infection in the production of the disease is overemphasized. He noted evidence of an intimate relationship between arthritis and the nervous system in the frequent occurrence of paresthesia and other vague nervous sensations. In all the large groups so

22. Walmsley, Thomas: J. Bone & Joint Surg. **10**:40 (Jan.) 1928.

23. Pemberton, Ralph: Michigan State M. J. **26**:599 (Oct.) 1927.

far studied, women predominated. In the group studied by the author, exposure played an important rôle. Anemia also occurred more frequently in the women who had arthritis. Pemberton's biochemical studies showed that the nitrogenous metabolism is normal in the disease; hence, there is no reason to restrict proteins in the diet. On the other hand, evidence of slowed sugar metabolism has been frequently noted, which the author explained on the basis of poor circulation with less oxygen available for the consumption of the sugar. The blood does not appear to reach the tissues adequately. In a study of the red blood count in arthritis, it was found that the first drop of blood had a lower count than the fourth or fifth, which is the reverse of normal. By studying the capillaries by Lombard's method, the author found them to be less well filled with blood than normal. The capillaries appeared to be contracted. Because of this observation the author experimented with the administration of sodium nitrate in persons with arthritis, but is not yet ready to evaluate the therapeutic efficiency of this measure. The author observed proliferative changes in the patellae of dogs after ligation of the blood vessels to the patella. Pemberton stated that with the exception of removal of foci of infection, vaccines and nonspecific protein therapy, the measures used achieve their effect through their influence on blood flow or metabolism, or both. Arsenic is of value in the treatment of the anemia. Colonic irrigation and colonic massage are sometimes helpful, but in the author's opinion regulation of what is taken into the mouth is of more value. He considered the development of the mechanistic conception of disease and orthopedic treatment based on it a distinct advance. In conclusion, the author attempted to answer the question of what avail is treatment. Of 188 patients who were followed, 24 per cent were completely cured and 73 per cent were definitely improved. He expressed the opinion that most of the present sufferers from arthritis could be saved for a useful life by the methods of treatment now at our command.

Schauffler,[24] writing on the subject of chronic arthritis, urged that every physician use some simple method of classification and familiarize himself with the major characteristics of each group in order that he might treat his patients to the best advantage. He has found the most serviceable clinical grouping to be that of Goldthwait: (1) infections; (2) atrophic; (3) hypertrophic arthritis. He stated his belief that the pure infectious type is always due to bacterial infection. The atrophic type is of a different character. The patients are usually of the visceroptotic type and have always been somewhat delicate. There is usually a history of worry, overwork or mental shock, and there is evidence of disturbed body chemistry or of endocrine imbalance.

24. Schauffler, Robert M.: Arthritis, J. A. M. A. **89**:1748 (Nov. 19) 1927.

Bacterial infection may have provided the final thrust which started the disease. In the author's opinion, such patients are never cured or the condition arrested by any single line of attack such as removal of foci of infection. Hypertrophic arthritis presents an almost opposite picture. The patients are usually of the thickset type. Bacterial infection is not the cause of the condition, and it cannot be cured by removal of the local foci. Schauffler expressed the view that in the infectious type removal of the foci of infection and the administration of iodoxybenzoate sodium or ammonium intravenously with careful technic is the most efficient treatment in subacute and chronic cases.

Etiology.—Cecil [25] discussed the etiology of chronic arthritis. His views may be summarized as follows: In infectious arthritis, the inciting cause is focal infection. The predisposing causes are old age, overweight, faulty posture, occupation, physical defects and exposure. In metabolic arthritis or gout, the exciting cause is uric acid. The predisposing cause is purine diet and heredity.

Jones [26] expressed the belief that the lesions in the joints of persons with osteo-arthritis are end-results which may be arrived at by a variety of pathologic states. Quantitative and qualitative alterations, in internal secretions led to disturbances in the normal current of metabolism. It is the opinion of the author that the disease is due to nutritional alterations in the articular structures. They are not inflammtory. Hypothyroidism is common in the disease. Trauma is often the inciting cause. There are two stages in the history of osteo-arthritis. The primary stage is marked by symptoms of villous overgrowth. The later or terminal is the osteo-arthritic phase. Static factors are important. The disease should be recognized in the initial stage. Roentgen-ray examination is usually negative in the early stage.

Relation of Surgical Pathologic Conditions of the Right Lower Quadrant to Chronic Arthritis.—Discussing the relationship of surgical pathologic conditions of the right lower quadrant of the abdomen to chronic arthritis, Taylor [27] described the lesions he had been able to demonstrate in patients affected with this disease by roentgen-ray examinations after the patient had ingested an opaque meal. The author's studies and observations are of interest in suggesting the amount of information that may be obtained in regard to the presence and causes of intestinal stasis by thorough work and enthusiastic cooperation between the roentgenologist and the clinician. They point to an association between intestinal conditions and polyarthritis.

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25. Cecil, R. L.: Southern M. J. **21**:20 (Jan.) 1928.
26. Jones, A. B.: M. J. & Rec. **126**: 57 (Nov. 2) 1927.
27. Taylor, Raymond G.: J. Bone & Joint Surg. **10**:62 (Jan.) 1928.

Smith [28] expressed the belief that intestinal stasis is responsible for many cases of polyarthritis, and that in certain cases surgical intervention to relieve the cause of it is required. However, this should never involve resection or sidetracking of the involved portion of the intestine. He also believed that implantation of *Bacillus acidophilus* in the cecum is helpful in certain cases.

[ED. NOTE.—Surgeons were at one time enthusiastic in the attempt to relieve persons with intestinal stasis by operations. Numerous operations were performed in different disease conditions, including arthritis. The results were disappointing, and the radical method of treatment has been generally abandoned. Smith obtained some interesting results, and his work is suggestive and should be continued. Its chief value at present, however, is again to call attention to the importance of the intestinal factor in the treatment of persons with arthritis. The present armamentarium of the physician is varied and large enough to deal with this condition without resorting to operation.]

Treatment of Arthritis with O-Iodoxybenzoic Acid.—Of twenty-one patients with chronic arthritis who were treated with salts of o-iodoxybenzoic acid, Cottrell [29] found that all but three showed improvement. The relief of pain was often prompt and marked. In the majority of chronic cases, there was an improvement of function from a slight to a marked degree. In the author's opinion the drug is best given in courses of from six to eight doses semiweekly, with from three to six weeks' rest between each course. Intravenous administration is the method of choice, although the drug may be given orally in capsule form. The latter may be followed by nausea.

Gout.—According to Harbinson,[30] gout is probably often overlooked. All parts of the body should be examined for tophi. It should be remembered that in this condition bursae often become inflamed. The diagnosis is made by examination of the tophi. The author places considerable reliance on the determination of blood uric acid and advises that this examination be made in every patient with obscure pains in the joints. A blood uric acid content of more than 5 mg. is suggestive of gout. Other conditions in which the blood uric acid may be high are leukemia, polycythemia, interstitial nephritis and febrile states. The treatment is dietary, with purines eliminated. The protein may be high. Colchicum is still the best drug to use.

Gonorrheal Arthritis.—Thomas [31] wrote that the incidence of arthritis as a metastatic complication of gonorrhea is not great, averaging

28. Smith, Rea: J. Bone & Joint Surg. **10**:57 (Jan.) 1928.
29. Cottrell, J. E.: Am. J. M. Sc. **174**:623 (Nov.) 1927.
30. Harbinson, J. E.: California & West. Med. **27**:352 (Sept.) 1927.
31. Thomas, B. A.: Gonorrheal Arthritis, J. A. M. A. **89**:2174 (Dec. 24) 1927.

only from 2 to 3 per cent. In a series of 107 cases, the distribution according to sex was males, 97; females, 10. Thomas believed that the precipitating factor is not infrequently trauma, either directly to the joint or indirectly in the form of ill advised or careless urethral instrumentation and treatment, excessive activity or sexual excitement during the acute stage of the urethritis. The arthritic symptoms usually manifest themselves during the second or third week; the earliest case recorded is five days. However, involvement of the joints may supervene at any time throughout the acute or chronic course of the disease or in the presence of urethral or uterine adnexal complications. In Thomas' experience, the symptoms have been polyarticular in 58 per cent and monarticular in 42 per cent. The joints have been involved in the following order of frequency: knee, 58 cases; hip, 50; wrist, 21; shoulder, 19; phalangeal, 17; elbow, 13; metatarsophalangeal, 8; spine, 8; metacarpophalangeal, 7; sacro-iliac, 1; temporomaxillary, 1, and sternoclavicular, 1. In respect to treatment, Thomas called attention to what he believes is a current mistake on the part of the orthopedic surgeon and the genito-urinary surgeon; namely, that they center too much on the treatment of the joint or the prostate or the seminal vesicles as the foci of infection, with apparent lack of appreciation of the fact that the condition is a septicemia. Thomas felt that the first step should be to treat the blood stream infection either by biologic therapy or by chemotherapy; second, to eradicate the focus or foci of infection in the genito-urinary tract, and third, to treat the involved joint or joints by local methods.

(To be Continued)

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